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CONTROL OF DIABETES MELLITUS WITH PROTAMINE ZINC INSULIN IN SURGERY*

(BASED UPON A STUDY OF 25 CASES)

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IN previous reports from this Clinic[1, 2] it was shown that protamine zinc insulin has a wide application in the treatment of diabetes mellitus. With the use of our high carbohydrate-low calorie diet by the *ladder* method, as described, protamine zinc insulin was found equally effective in a variety of associated conditions and complications, with and without infections and with and without acidosis.[2] The results reported were largely those obtained while the patients were in the hospital. The subsequent data, however, in these cases, to date at least, show that these results were not temporary. One of the characteristic effects of protamine zinc insulin, judging from the subsequent reductions of dosages and changes of diet in these cases, appears to be rapid improvement in carbohydrate tolerance.

In order to evaluate protamine zinc insulin properly, surgical conditions were purposely excluded from the above studies, because of the many variables which may account for irregular fluctuations of dosages, irrespective of the type of insulin used. Anæsthesia, surgical trauma, and post-operative dietary difficulties are examples. Another condition which may add to the difficulties of interpretation of insulin dosages in surgery is the use of drugs which, because of their hyperglycæmic action, tend to counteract the action of insulin — epinephrin, pituitrin, morphine, etc. The use of protamine zinc insulin in such cases was, therefore, regarded as proper subject matter of a separate investigation.

One of the great advantages of protamine zinc insulin is the smaller number of injections necessary to control the diabetes. In order, however, that it may be applicable to surgery, protamine zinc insulin must also possess the following attributes:

1. *Rapid action,* for preparation of the diabetic for emergency operations, and

2. *Sustained action,* (*a*) to combat possible post-operative disturbances of carbohydrate metabolism due to the trauma or anæsthesia; (*b*) to permit the use of intravenous injections of glucose whenever necessary; and (*c*) to prevent delayed healing of tissues.

RAPID ACTION OF PROTAMINE ZINC INSULIN

In Charts 1, 2 and 4 of one of our previous reports[2] it was shown that, though the rate at which the blood sugar decreases with protamine zinc insulin is, as a rule, slower than with unmodified insulin, the blood sugar may be made to decrease very rapidly with protamine zinc insulin, providing the dose is large. These subjects, however, were uncomplicated diabetics. That the blood sugar may be made to decrease as rapidly with protamine zinc insulin as with unmodified insulin, even in the presence of severe infections, is clearly shown in Chart 1. It will be noted that of the two patients with carbuncles one was in a state of pre-coma, and that, of the two with gangrene of the extremities, one also suffered from severe acidosis. These 4 patients, therefore, all had infection—a condition known to interfere at times with the action of insulin, and 2 of the 4 had severe acidosis—another condition which may, at times, demand very large dosages of the unmodified insulin.

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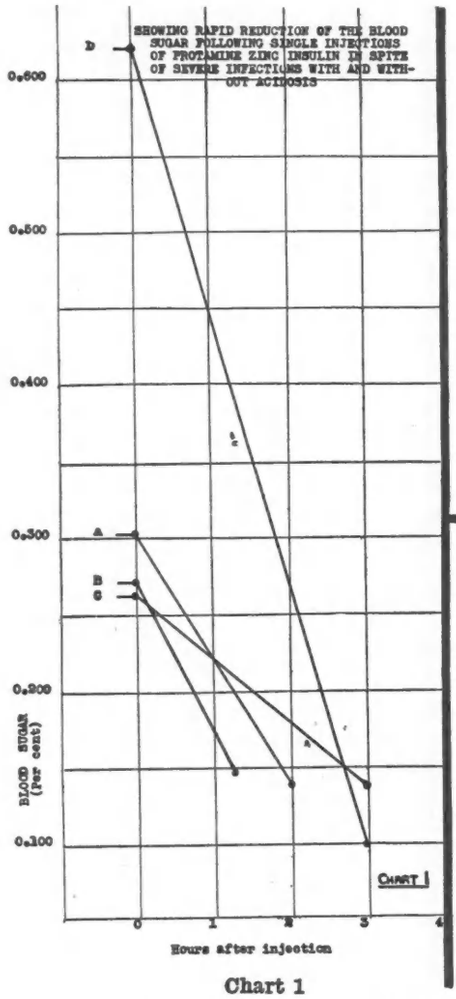
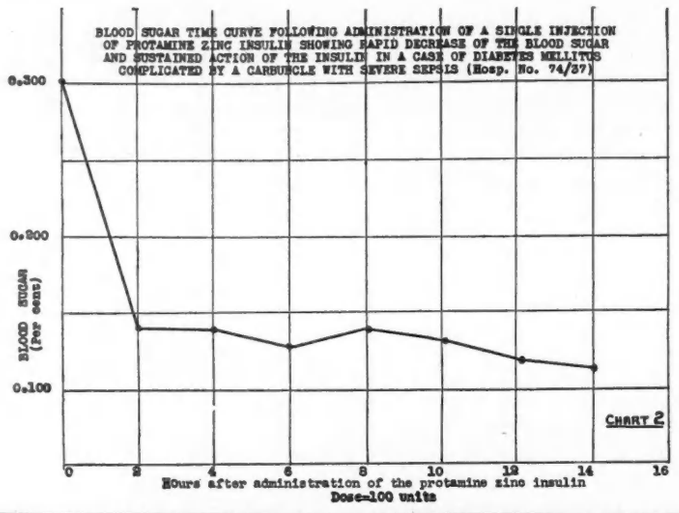
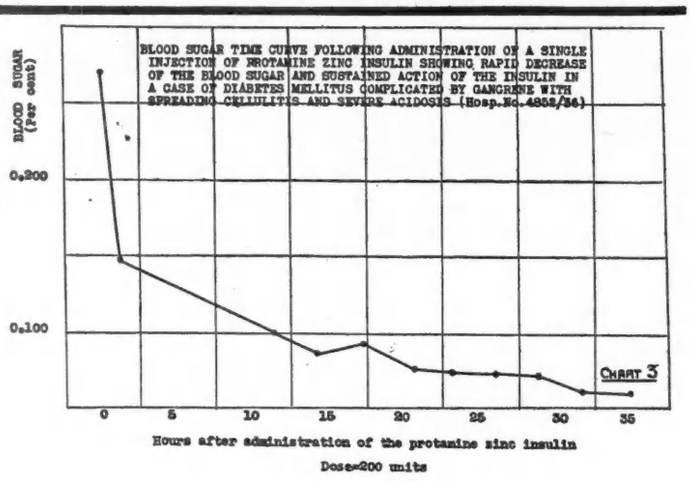



BLOOD SUGAR TIME CURVE FOLLOWING ADMINISTRATION OF A SINGLE INJECTION OF PROTAMINE ZINC INSULIN SHOWING RAPID DECREASE OF THE BLOOD SUGAR AND SUSTAINED ACTION OF THE INSULIN IN A CASE OF DIABETES MELLITUS COMPLICATED BY A CARBUNCLE WITH SEVERE SEPSIS (Hosp. No. 74/37)
0.300
0.200
0.100
BLOOD SUGAR (Per cent)
CHART 2
0 2 4 6 8 10 12 14 16
Hours after administration of the protamine zinc insulin
Dose=100 units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0.200
0.100
BLOOD SUGAR (Per cent)
CHART 3
0 5 10 15 20 25 30 35
Hours after administration of the protamine zinc insulin
Dose=200 units

Chart 1

A — 74/37 Carbuncle. No acidosis ..... 100 units
B — 4852/36 Gangrene. Spreading cellulitis. Pre-coma ..... 200 "
C — 159/37 Carbuncle. Pre-coma ..... 200 "
D — 1245/37 Gangrene. Spreading cellulitis. No acidosis.... 200 "

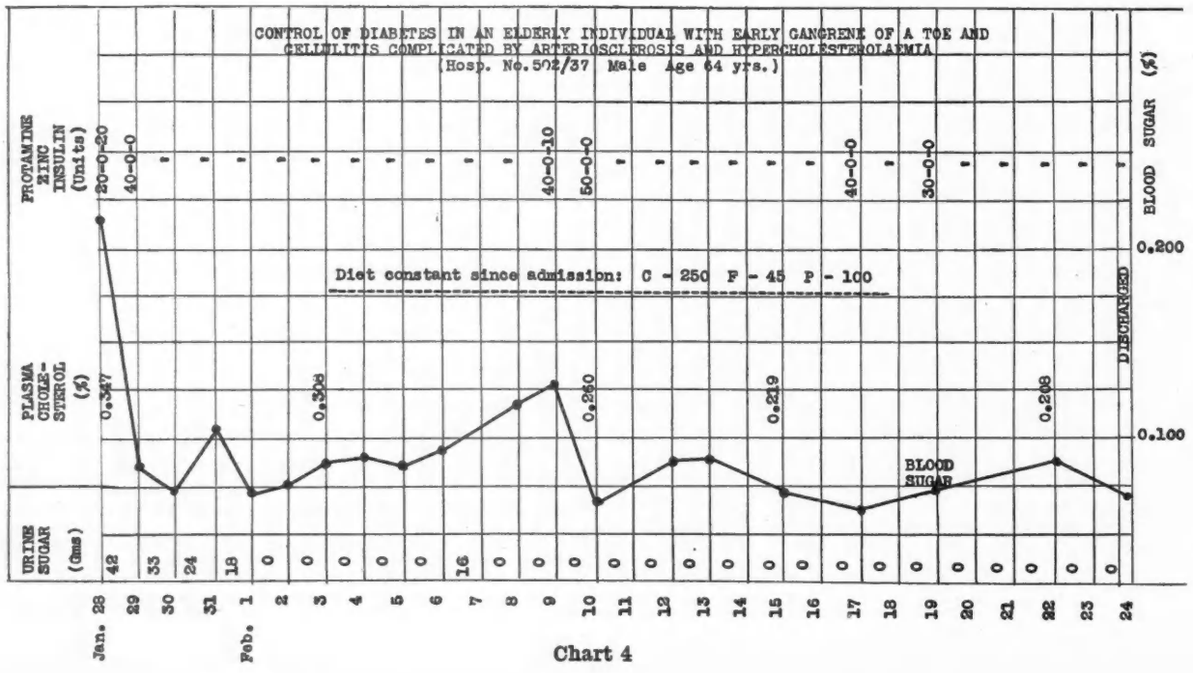
CONTROL OF DIABETES IN AN ELDERLY INDIVIDUAL WITH EARLY GANGRENE OF A TOE AND CELLULITIS COMPLICATED BY ARTERIOSCLEROSIS AND HYPERCHOLESTEROLAEMIA
(Hosp. No. 502/37 Male Age 64 yrs.)
PROTAMINE ZINC INSULIN (Units)
20-0-20
40-0-0
40-0-10
50-0-0
40-0-0
30-0-0
Diet constant since admission: C - 250 F - 45 P - 100
PLASMA CHOLESTEROL (%)
0.347
0.306
0.260
0.219
0.208
BLOOD SUGAR
BLOOD SUGAR (%)
0.200
0.100
DISCHARGED
URINE SUGAR (Gms)
42 33 24 18 0 0 0 0 0 16 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
Jan. 28 29 30 31 Feb. 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24

Chart 4

In spite of these conditions it will be noted that the blood sugar was rapidly reduced to the normal or nearly normal level in every case. Thus:

TABLE I.

Hospital No.	Condition	Acidosis	Protamine zinc insulin (units)	Blood sugar (per cent)		
				Before injection	After injection	Time (minutes)
74/37	Carbuncle	Absent	100	0.302	0.137	120
4852/36	Gangrene	Pre-coma	200	0.270	0.149	75
159/37	Carbuncle	" "	200	0.256	0.137	180
1245/37	Gangrene	Absent	200	0.625	0.094	180

The above cases were purposely selected because of the severity of the infections. It is obvious, therefore, that as far as infection is concerned protamine zinc insulin is not contraindicated in surgery. Parenthetically, it may here be pointed out that when such large doses as shown in the above four cases are given it is our practice to inject the suspension at three or four different sites of the body, not only to avoid the discomfort of distension of the tissues due to bulk but also to insure more rapid absorption.

SUSTAINED ACTION OF PROTAMINE ZINC INSULIN

As stated previously, in order that protamine zinc insulin may be applicable to surgery its action must be sufficiently prolonged to combat possible post-operative complications, to permit intravenous injections of glucose, and to prevent delayed healing of tissues. That the action of protamine zinc insulin may be prolonged, even in the presence of severe infections, is clearly shown in Charts 2 and 3. In Chart 2 is the record of a blood sugar time curve obtained in one of the above-mentioned cases of carbuncle (Hosp. No. 74/37), and in Chart 3 is recorded a similar curve obtained in one of the above-mentioned cases of gangrene with spreading cellulitis and acidosis (Hosp. No. 4852/36). In each case the curve was obtained after a *single* injection of protamine zinc insulin. It will be noted that the blood sugar was still normal 14 hours after the injection in one case and 35 hours after in the other. Better proof, however, of the applicability of protamine zinc insulin to surgery, so far as sustained action is concerned, will be seen in the control of the diabetes (*a*) immediately after operation, (*b*) during intravenous injection of glucose, (*c*) in post-operative healing of wounds, and (*d*) in the recovery of patients from surgical conditions without operation.

THE SELECTION OF CASES

In order to evaluate the action of protamine zinc insulin with respect to control of the diabetes immediately after operation an attempt was made to select a variety of cases with regard to type of anæsthetic, duration of anæsthesia, type of operation, and severity of the diabetes. In Table II are recorded the 25 cases selected for this purpose, and it will be noted that very few of these conditions could be regarded properly as minor; amongst them there were 5 cases of gangrene of the extremities, 4 of cellulitis of the feet and 2 of carbuncle. In Table III are recorded the 14 operations which were necessary in these cases, and it will be observed that they included 2 cholecystectomies, 1 hysterectomy, and 2 amputations at the thigh. The types of anæsthesias and their durations are also recorded in the same Table. Of special interest are blood-sugar determinations before and one hour after operation in each case.

TABLE II.

SHOWING CONDITIONS OF SURGICAL PATIENTS IN WHOM THE DIABETES WAS CONTROLLED WITH PROTAMINE ZINC INSULIN ONLY

Diagnosis*	Number
Abscess—foot	2
peri-anal	1
tooth	2
Birth trauma	1
Carbuncle	2
Cellulitis, foot	4
Cholecystitis, chronic	2
Fracture, humerus	1
Gangrene, foot	5
Mastoiditis, acute	1
Ovarian cyst	1
Pregnancy	1
Pyelitis	1
Tonsillitis	1

*For operations for these conditions see Table III.

TABLE III.

SHOWING DEGREE OF CONTROL OF DIABETES ONE HOUR AFTER OPERATION WITH PROTAMINE ZINC INSULIN

Hospital No.	*Operation*	*Anæsthetic*		*Blood sugar (per cent)*		*Protamine zinc insulin*	
		Type	*Duration (min.)*	*Before*	*1 Hour after*	*Units*	*Time of injection before operation (minutes)*
4852/36	Incision of abscess	Nitrous oxide	15	0.156	0.175	40	165
"	" " "	" "	15	0.095	0.204	60	165
"	Amputation at thigh	Spinal		0.113	0.126	100	30
5820/36	" " "	Nitrous oxide	45	0.100	0.072	60	290
6512/36	Tonsillectomy	Local		0.087	0.131	40	215
6558/36	Amputation, toe	Nitrous oxide	20	0.114	0.100	70	325
"	Incision of abscess	" "	10	0.128	0.122	30	210
74/37	Carbuncle	" "	10	0.137	0.137	100	195
"	"	" "	10	0.232	0.137	100	500
159/37	"	" "	30	0.060	0.048	100	195
730/37	Cholecystectomy	" "	150	0.135	0.149	10	150
734/37	"	Ether	90	0.057	0.196	30	1645
770/37	Hysterectomy*	"	90	0.166	0.153	40	170
"	Repair of birth trauma	Spinal		0.137	0.086	50	180

*Also removal of ovarian cyst.

THE CONTROL OF BLOOD SUGAR ONE HOUR AFTER OPERATION BY THE PRE-OPERATIVE INJECTION OF PROTAMINE ZINC INSULIN

During the last fifteen years it has been a routine practice in this Clinic to determine the blood sugar in surgical cases before and, again, exactly one hour after the operation, in order to detect possible harmful effects of the anæsthesia or operation, and so permit the institution of the necessary treatment for the diabetes with as little delay as possible. This was also the practice with the cases treated with protamine zinc insulin.

To compare the results of protamine zinc insulin with those obtainable with the unmodified insulin a similar number of cases treated with the latter type of insulin were selected from our records. The selection was strictly random, except that, in order that the data may be reasonably comparable, an attempt was made to approximate conditions of the two groups of cases as closely as possible with respect to (*a*) type of anæsthetic; (*b*) duration of anæsthesia, and (*c*) type of operation. The data of the unmodified insulin are shown in Table IV. In both Tables III and IV are also recorded the

TABLE IV.

SHOWING DEGREE OF CONTROL OF DIABETES ONE HOUR AFTER OPERATION WITH UNMODIFIED INSULIN

Hospital No.	*Operation*	*Anæsthetic*		*Blood sugar (per cent)*		*Unmodified insulin*	
		Type	*Duration (min.)*	*Before*	*1 Hour after*	*Units*	*Time of injection before operation (minutes)*
6047/35	Incision of abscess	Nitrous oxide	15	0.105	0.169	10	135
4211/34	" " "	" "	15	0.370	0.200	10	375
569/36	Amputation, thigh	Spinal		0.117	0.149	10	150
602/32	" "	Nitrous oxide	50	0.161	0.131	10	180
3024/36	Tonsillectomy	Local		0.181	0.277	10	270
7236/29	Amputation, toe	Nitrous oxide	20	0.263	0.270	20	210
1746/30	Incision of abscess	" "	10	0.256	0.186	10	15
5699/26	Carbuncle	" "	10	0.232	0.220	30	90
5525/24	"	" "	15	0.074	0.109	30	210
2644/35	"	" "	20	0.128	0.139	20	300
3874/35	Cholecystectomy	" "	100	0.117	0.208	5	330
2704/31	"	Ether	75	0.156	0.322	15	240
7249/30	Hysterectomy*	"	100	0.217	0.344	15	30
3521/35	Repair of birth trauma	Spinal		0.153	0.181	5	60

*Also removal of ovarian cyst.

number of units of insulin administered in each case before operation and the interval between the time of injection and the time of operation. The combined data of both Tables III and IV are briefly summarized in Table V.

TABLE V.

THE DEGREE OF CONTROL OF DIABETES ONE HOUR AFTER OPERATION BY THE PRE-OPERATIVE ADMINISTRATION OF UNMODIFIED INSULIN AND PROTAMINE ZINC INSULIN

	Insulin					*Average blood sugar (per cent)*		
			On day of operation					
Number of cases	*Type*	*Day before operation (units)*	*Units before operation*	*Average time of injection before operation (minutes)*	*Total units during day*	*Before operation*	*After operation*	*Increase*
14	Unmodified	36	14	185	41	0.181	0.207	0.026
14	Protamine zinc	75	60	317	71	0.123	0.131	0.008

It will be observed at once that the average control of the diabetes both before and after the operations was much better with the protamine zinc insulin than with the unmodified insulin. With the unmodified insulin the average blood sugars before and after operation were 0.181 and 0.207 per cent, respectively, with an average post-operative increase of 0.026 per cent, whereas with the protamine zinc insulin the average blood sugars before and after operation were 0.123 and 0.131 per cent, respectively, with an average post-operative increase of 0.008 per cent only.

There are a number of difficulties in the interpretation of the above data. Though the two groups were approximately alike with respect to type of operation, type of anæsthetic, and duration of anæsthesia, an important variable, and one very difficult to control in the selection of cases for comparison, was the degree of severity of the diabetes. It will be noted (Table V) that the average pre-operative dose of unmodified insulin was about one-quarter of that of the protamine zinc insulin. This alone, however, is no indication of the severities of the diabetes in the two groups, since the methods of treatment were not the same. With the protamine zinc insulin the diabetes was treated in the majority of cases with single daily injections, whereas with the unmodified insulin the total daily requirements were divided into two, three, and in some cases four or more injections, in accord with past experiences that small amounts of unmodified insulin at frequent intervals were more effective than large single injections. That the average severity of the diabetes in the group of cases treated with the unmodified insulin was less than in the group treated with the protamine zinc insulin is however suggested not only from the clinical data in general but, also, from the total insulin requirements the *day before operation*. It will be noted that whereas the average requirement of protamine zinc insulin was 75 units that of the unmodified produet was 36 units only.

Assuming that the average severity of the diabetes was less in the group of cases treated with the unmodified than in the group treated with the protamine zinc insulin, one would expect *a priori* that the degree of the control of the diabetes both before and immediately after operation would have been better with the unmodified type. Actually, it will be observed, judging from the blood sugars, it was not so, and the explanation appears to be the relatively transient action of the unmodified insulin. It should be noted that the blood sugars before operation were obtained in the fasting state and immediately before the injection of the insulin. In the case of the protamine zinc insulin they were, therefore, obtained in the majority of cases 24 hours after the last injection; whereas, with the unmodified insulin, because of the practice of more frequent injections, they were obtained, in the majority of cases either 6 or 8 hours only after the last injection. In the interpretation of the post-operative blood sugars it should also be noted that, though the average time allowed for the insulin to act was not the same in both groups of cases, the periods corresponded to the averages necessary to obtain

the maximum results, namely, about 3 hours with the unmodified insulin and about 5 hours with the protamine zinc insulin. It is also of interest to note that, whereas the average amount of insulin required during the *entire* day of the operation was slightly less than the day before in the case of the protamine zinc insulin, it was slightly more in the group treated with the unmodified insulin. It is quite possible, however, that, in spite of all of the above efforts to control conditions, the data of the two groups may not be strictly comparable. The fact of practical importance (and clearly shown in Table V) is that protamine zinc insulin is not only not contraindicated in the preparation of patients before operation but is definitely indicated.

THE CONTROL OF DIABETES WITH PROTAMINE ZINC INSULIN FOLLOWING THE INTRAVENOUS INJECTION OF GLUCOSE

Injection of glucose solutions intravenously is a common practice in surgery immediately and, at times, for some days after operation. To be effective, that is, to supply food, combat acidosis, etc., the injected glucose must be utilized. Even in the very mild diabetic, however, operation and anæsthesia of long duration tend to disturb the carbohydrate tolerance sufficiently to prevent complete utilization. Glycosuria is a frequent finding following such injections. In Table VI are, therefore, recorded the data obtained in one of the above-mentioned cases. This patient was purposely selected because of (*a*) the known severity of the diabetes (known carbohydrate tolerance and requirements of the unmodified insulin) for 3 years before the unmodified zinc insulin was replaced by the protamine zinc insulin;* (*b*) the known protamine zinc insulin requirements for three months before the operation;* (*c*) the major type of the operation (cholecystectomy); (*d*) the type of anæsthesia (ether) and (*e*) its long duration (90 minutes).

TABLE VI.

THE CONTROL OF DIABETES FOLLOWING INTRAVENOUS INJECTION OF GLUCOSE SOLUTION BY ADMINISTRATION OF PROTAMINE ZINC INSULIN 21 HOURS, 25 MINUTES BEFORE INJECTION

(Showing complete utilization of the injected glucose)
(Hosp. No. 734/37)

Date	*Hour*	*Urine sugar (Gms.)*	*Blood sugar (%)*	*Remarks*
1937 Mar. 4	8.00 a.m.		0.057	24 hours after 30 units protamine zinc insulin.
"	11.20 a.m.			Ether anæsthesia. Cholecystectomy.
"	1.50 p.m.		0.196	One hour after 90 min. ether anæsthesia.
"	2.20 p.m.			30 units protamine zinc insulin.
" 5	8.00 a.m.	None*	0.072	
"	11.45 a.m.			750 c.c. 5% glucose solution intravenously.
"	3.45 p.m.		0.200	20 units protamine zinc insulin.
" 6	8.00 a.m.	Trace†	0.156	

*Includes all urine of previous 24 hours.
†Includes all urine since injection of glucose.

It is a well known fact from renal threshold studies that there may be an appreciable lag between the injection of glucose and its appearance in the urine. To exclude this possibility in the interpretation of our results the urine of the 24 hours before the injection was compared with that collected 24 hours after the injection. A trace of sugar only, that is, less than 1 gram, was found in the latter specimen. It is obvious, therefore, that the glucose injected intravenously in this case was completely utilized. It will be noted that the blood sugar in this case one hour after operation (Table III) was 0.196 per cent, and that 4 hours after the injection of the glucose it was 0.2 per cent. A raised renal threshold for glucose, therefore, probably accounted to some extent for the failure of some of the injected glucose to appear in the urine.

THE RATE OF HEALING OF POST-OPERATIVE WOUNDS

As stated, one of the reasons for the need of sustained action of the protamine zinc insulin is to prevent delayed post-operative healing of tissues. In order, therefore, to evaluate protamine zinc insulin with respect to this phase of surgery, the cases were divided into two groups, namely, (*a*) those with clean abdominal wounds and (*b*) cases with infection.

Cases with no infections.—In Table VII are recorded the results noted in 3 major operations with clean abdominal wounds. The data require little comment. It will be noted that with re-

* The daily dosage of unmodified insulin from September, 1933, to December, 1936, was 30 units divided into 3 injections. The daily dosage of protamine zinc insulin from December, 1936, to the time of operation (March 4, 1937) was also 30 units, but given in single injections. No changes were made in the diet when the unmodified insulin was replaced by the protamine zinc insulin.

spect to the number of days after operation when (*a*) the clips were removed; when (*b*) the sutures were removed; when (*c*) the patients were allowed out of bed; and when (*d*) they were discharged from hospital the post-operative courses of these diabetics did not differ from those of non-diabetics with similar surgical conditions and exposed to similar types of operations.

Cases with infection.—That tissues of the diabetics may be made to heal well with protamine zinc insulin even in cases of infection is shown by a comparison of Tables II and III. It will be noted, for example, that all 4 patients with cellulitis of the feet and 2 of the 5 patients with gangrene recovered without operation. These 6 cases are of special interest because of the associated conditions, each of which *per se* may account for slow healing, that is, infection, age, duration of the diabetes, and the presence of arteriosclerosis. All were past middle age, one had no pulsations in the dorsalis pedis artery of the affected foot, 3 had no pulsations in the posterior tibial artery, and in 2 cases the pulsations were absent in both arteries. Space does not permit a detailed analysis of these cases. In Chart 4, however, are recorded the findings in the *severest* of these 6 cases (Hosp. No. 502/37).

TABLE VII.

SHOWING NORMAL RATE OF HEALING OF POST-OPERATIVE ABDOMINAL WOUNDS IN DIABETICS CONTROLLED BY SINGLE DAILY INJECTIONS OF PROTAMINE ZINC INSULIN

Hospital No.	Operation	Anæsthesia		Blood sugar (per cent)		Post-operative course (Number of days after operation)			
		Type	Duration (minutes)	Before operation	One hour after operation	Clips removed	Sutures removed	Up in chair	Discharged
6327/36	Hysterotomy	Spinal		0.076	0.059	5	10	12	13
734/37	Cholecystectomy	Ether	90	0.057	0.196	5	11	16	20
770/37	Hysterectomy	"	90	0.166	0.153	5	10	19	23*

*Operation for repair of birth trauma 23 days after hysterectomy.

CASE REPORT

A male, aged 64, was admitted to the hospital with a history of discoloration and throbbing pain in the small toe of the right foot of one month's duration. Clinically, there was evidence of generalized arteriosclerosis and the local condition was that of early gangrene with a slight degree of cellulitis which had spread to the dorsum of the foot. In addition also to the hyperglycæmia and glycosuria, the cholesterol content of the blood plasma was increased, namely, 0.347 per cent. It will be noted, however, that with a single daily injection of protamine zinc insulin the diabetes was brought under control rapidly and kept under control during the entire period of observation, and that 31 days after admission to the hospital the patient was discharged cured, as far as the local condition was concerned. The plasma cholesterol had decreased from 0.347 to 0.208 per cent.

It is a well known fact that infection of a foot may *per se* cause temporary absence of pulsations of the dorsalis pedis or posterior tibial artery. It is, therefore, necessary in such cases, from the point of view of prognosis, to differentiate this form of absence of pulsations from the permanent form due to arteriosclerotic changes. It is, therefore, of interest to note that in this case *both* feet were equally cold; both had no pulsations in the posterior tibial artery; and both had slight pulsations only in the dorsalis pedis. In this case, therefore, the control of the diabetes was such that perfect healing was obtained in spite of the marked degree of vascular occlusion.

Of interest also is the case (Hosp. No. 454/37) in which, following control of the diabetes, the acute mastoiditis disappeared without operation, except for puncture of the ear drum.

URTICARIA WITH PROTAMINE ZINC INSULIN

The following is our first case of urticaria met with in treatment with protamine zinc insulin.

CASE REPORT

The patient (Hosp. No. 730/37) was a male, aged 43, and a diabetic of 18 months' duration. He was admitted to the hospital for a cholecystectomy, and the diabetes was well controlled by single daily injections of protamine zinc insulin of from 10 to 40 units for ten days. On the eleventh day he complained of slight itchiness of the skin at the site of injection. The following day an urticarial rash appeared over both upper extremities. Sensitivity tests with one-half unit doses showed that he was sensitive to the unmodified (beef) insulin as well as to the protamine zinc insulin. Of interest, and fitting in with the prolonged action of protamine zinc insulin, is the fact that though the protamine zinc insulin was discontinued the urticaria became more generalized and persisted for three and one-half days.

With the unmodified insulin, with extremely few exceptions, the urticarial lesions in our cases have disappeared within 24 hours after the injections were discontinued. This case, therefore, is of interest not only because of the fact that it is the first we have met with but because of the long duration of the urticarial lesions.

SUMMARY

Though the action of protamine zinc insulin upon the blood sugar is ordinarily slower than that of the unmodified insulin, by the use of large injections it may be made to act as rapidly as the unmodified product. Therefore, with respect to rapid action alone protamine zinc insulin is not contraindicated in preparation of diabetics for operation nor for control of post-operative emergencies.

By proper dosage the action of protamine zinc insulin may be prolonged sufficiently by single daily injections to permit the use of intravenous injection of glucose after operations and, also, the normal rate of healing of wounds. The sustained action also appears to be sufficient to permit healing of infected tissues (cellulitis, etc.). In this study the cellulitis in 4 cases and the early gangrene with cellulitis in 2 cases disappeared without operation by control of the diabetes with single daily injections.

A case of urticaria is reported because it is the first met with in this Clinic with protamine zinc insulin, and also because of its long duration.

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SPONTANEOUS SUBARACHNOID HÆMORRHAGE AND BRAIN TUMOUR

(A REPORT OF 3 CASES)

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THE syndrome of spontaneous subarachnoid hæmorrhage has only recently received general recognition as a clinical entity. It may be produced by a variety of etiological factors. Symonds,[1] whose classical article in 1924 stimulated modern clinical interest in this condition, collected 124 cases from the literature and added 3 more of his own. In a critical analysis he found that by far the largest group were those caused by arteriosclerosis and aneurysm. In 41 of these cases no satisfactory cause could be found, despite the fact that 18 of them came to autopsy. So many comprehensive accounts have since appeared on the subject that it is unnecessary to undertake a review of the literature here. Interest in the condition arises from the importance of an early diagnosis and the fact that once the condition is recognized, proper treatment instituted early yields excellent results.

In 1933 one of us (C.K.R.)[2] reported a series of 26 cases which were analyzed from the standpoint of their etiology, symptomatology and clinical course. In most of these cases developmental defects in the walls of the cerebral arteries were the underlying etiological factors. This was in agreement with the work of Forbus,[3] who demonstrated a muscular deficiency at bifurcations along the course of the circle of Willis. It is at or close to these points that aneurysms occur and subsequent hæmorrhage. Other cases were due to atheromatous changes in the vasculature. In a surprising number of cases spontaneous subarachnoid hæmorrhage occurred as a complication of the various stages of pregnancy. Lues is rarely a factor.

Dowling[4] (1933) proposed what seems to be the simplest and most comprehensive etiological classification as follows.

1. Blood dyscrasias.
2. Extrinsic diseases affecting the blood vessels of the subarachnoid space (*i.e.*, meningitis, lues, embolism and heat stroke).

* From the Montreal Neurological Institute.

3. Intrinsic abnormalities of the blood vessels (*i.e.*, thromboses, degeneration of the blood vessel wall—arteriosclerotic, hypertensive, toxic and infective—congenital nævi, and congenital aneurysms).

Brain tumour is not mentioned specifically as a cause.

That brain tumours may cause bleeding into the subarachnoid space, either primarily, or secondarily from the ventricles, has been tacitly accepted. But it is extremely rare to find specific cases cited in the literature in which, pre-operatively, brain tumours have given rise to the syndrome of subarachnoid hæmorrhage. In Symonds' study tumours and cysts were grouped together and his analysis showed them to be an infrequent cause. Of his 127 cases there were only 2 that fell into this category. In one there was an old cyst which was thought to be a relic of a meningo-encephalitic infection 10 years previously, and a vessel in the wall of this cyst had ruptured directly into one of the lateral ventricles; the other was a case of cyst-formation following head injury, with eventual rupture of a vessel in its wall. Neither of these, of course, is a true neoplasm, and evidence that cerebral tumour causes massive subarachnoid hæmorrhage is extremely rare in the literature.

Since 1930 more than 200 cases of spontaneous subarachnoid hæmorrhage have been reported. In reviewing them, we found that in only one case, that of Globus, Strauss and Ginsberg,[5] was a glioma recorded as the etiological factor. Their patient presented signs of a bilateral cerebellar lesion and later developed a left hemiparesis and bilateral 7th nerve paralysis. In this respect he differs from the patients reported in this article, none of whom presented any demonstrable localizing signs. Globus, Strauss and Ginsberg stress the fact that in their collection of 150 verified tumours of the brain this was the only one in which tumour gave rise to subarachnoid bleeding during the pre-operative course, and they comment further that hæmorrhage into the subarachnoid space from a bleeding tumour of the brain is rare.

Oldberg,[6] reviewing 832 cases of glioma from Cushing's clinic, found that spontaneous massive hæmorrhage had occurred in only 32, or 3.7 per cent. In these the hæmorrhage was accompanied by appropriate clinical symptoms in only 7, or 0.84 per cent. So it can be said that in only the latter percentage of these cases did the patient present a picture simulating subarachnoid hæmorrhage. He adds, moreover, that the same type of symptoms may occur in gliomata with no hæmorrhage—due to occlusion of the cerebral blood vessels, shutting off the foramen of Munro or aqueduct of Sylvius, etc.

Recently in our clinic, Elvidge, Cone and Penfield[7] reviewed 220 consecutive cases of glioma, and in none of these did the clinical picture at any time before operation resemble that of spontaneous subarachnoid hæmorrhage. Many additional cases of glioma could be added since this review, but 2 of the cases reported here are the first to present such a picture.

Schultz[8] (1935) presented 3 cases in which there was massive hæmorrhage into previously unrecognized gliomata of the brain, but they are not reported fully enough to determine whether blood was found by lumbar puncture.

Turning to the blood vessel tumours of the brain, despite what one would expect, the syndrome is also comparatively infrequent. Unfortunately, as will be discussed more fully at the end of Case 3, there is a certain amount of confusion in the literature as regards the classification and nomenclature of aneurysms and angiomata. The term aneurysm should be retained for simple saccular expansions of a blood vessel, and these of course are among the most common causes of spontaneous subarachnoid hæmorrhage. The blood vessel tumours which consist of angiomas and hæmangiomas give rise to this syndrome much more infrequently. In Bailey and Cushing's monograph[9] on this subject, of the 25 cases reported, only 2 (Nos. 12 and 19) gave a history suggestive of subarachnoid hæmorrhage. Bergstrand, Olivecrona and Tönnis[10] reported 3 such cases in their recent book on vascular malformations and tumours of the brain. Frank[11] described two cases of hæmangioma in which pre-operatively blood was found in the spinal fluid. In his article on blood in the cerebrospinal fluid Bagley[12] presented a series of 27 cases, and of these there was one in which the hæmorrhage was proved to be due to tumour. This was in an infant aged 10 days with a neoplasm of the choroid plexus which bled into the lateral ventricle.

The present report deals with 3 unusual cases of cerebral tumour which produced the syndrome of subarachnoid hæmorrhage. They are interesting because of their comparative rarity, and are presented to supplement the list of verified etiological factors in this syndrome.

CASE 1

H.S., male, aged 22, a patient of Dr. J. N. Petersen, was admitted to the Montreal Neurological Institute on July 25, 1935, with the following history. Headache had been present on and off for the past month. This headache was mainly frontal, dull, occasionally throbbing and occasionally accompanied by pain over the eyeballs, especially the right. It was present in the morning on arising and gradually wore off during the day. For the past six days, however, it had been present constantly, and there was some photophobia. For the past five days there had been dizziness and constipation, and for three days he had vomited a few minutes after every meal.

The family and past personal history were irrelevant. At the time of admission, on examination, the patient was complaining of right frontal headache and photophobia. His chest, back and forearms were covered with a brownish, finely pigmented scaly rash, typical of pityriasis versicolor. The head, ears, nose and sinuses were normal. There was slight tenderness over both eyeballs. The tongue was reddened and coated. His respiratory and cardiovascular systems were normal. Blood pressure, 110/76. The extremities were normal. Neurological examination was normal in all respects, except that the upper abdominal reflexes on both sides could not be obtained. There was no stiffness of the neck or positive Kernig sign. Some of his mental reactions were unusual but insufficient to label definitely. No cranial bruit was audible.

Laboratory data.—Urine (on admission), normal. *Blood count* (on admission): 4,500,000 red blood cells; 15,000 white blood cells; 95 per cent hgb. Differential count showed an increase in immature polymorphonuclear cells, with 80 per cent of polymorphonuclears.

X-rays of skull.—Antero-posterior and stereo right lateral views showed a slightly thickened vault, with no evidence of increased intracranial pressure. The pineal gland was calcified and not displaced, as seen in the lateral views. X-rays of the accessory sinuses of the nose were normal.

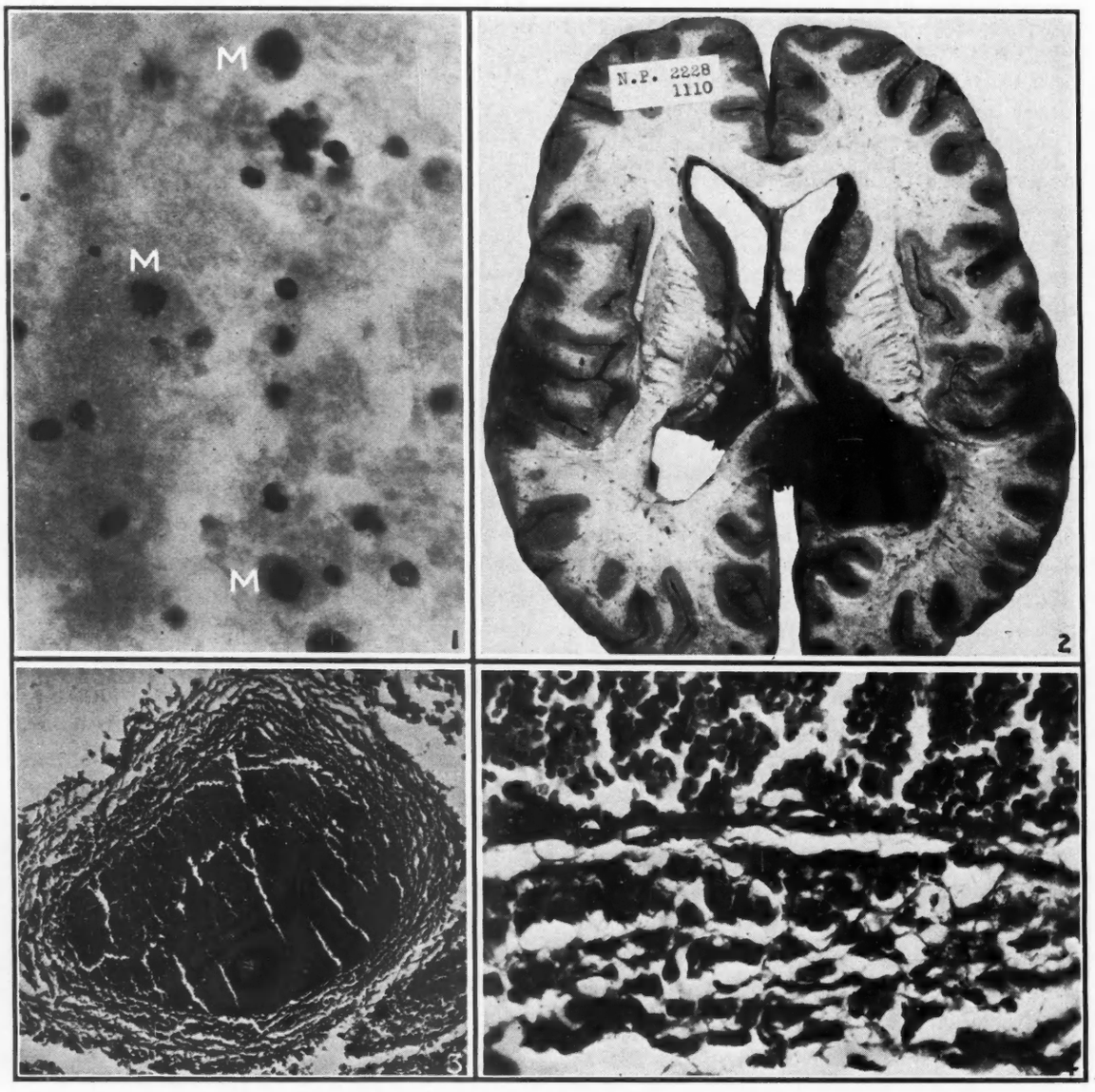

Fig. 1.—Mitotic division (M) of large mononuclear cells in the spinal fluid of Case 1. **Fig. 2.**—Case 1. Horizontal section of brain showing tumour occupying the right occipital region, in the posterior part of the ventricle, and invading the splenium of the corpus callosum. **Fig. 3.**—Case 1. Branch of the posterior cerebral artery surrounded by a thick perivascular collar of tumour cells. **Fig. 4.**—Case 1. Higher magnification of part of the vessel wall showing invasion of tumour cells up to the endothelium and obliteration of the normal structure of the vessel wall.

Lumbar puncture.—(On admission) initial pressure 140 mm. of water (recumbent); the fluid was of a very faint lemon yellow colour; 430 red blood cells, 4 white blood cells; negative Pandy; proteins, 45.7 mg. per cent.

Ophthalmological consultation.—On admission, optic discs normal (Dr. Tooke).

During his first 10 days in hospital the spinal fluid became clearer, and on August 9th there were only 226 red blood cells but the cerebrospinal pressure had increased to 285 mm. of water. On one or two occasions there was transient stiffness of the neck and a questionably positive Kernig sign on the left side. On August 9th the disc margins were not as clear cut as on admission. Two days later there was a sudden increase in the occipital pain, rigidity of the neck and the Kernig sign, and on lumbar puncture the pressure was 400, the fluid bloody, and contained 77,000 red blood cells per c.mm. with a positive Pandy reaction. There were no other positive physical signs, and a diagnosis of spontaneous subarachnoid hæmorrhage was made on the basis of this general clinical picture. From then on the spinal fluid never became clear. The patient constantly complained of severe continual headache with intense exacerbations, and there was increasing restlessness and neck rigidity. Symptomatic relief could be obtained only by repeated lumbar puncture. In all, 76 punctures were done, each followed by some improvement except towards the terminal phases of the disease. The acute exacerbations were characterized by a definite large increase in the number of red cells in the spinal fluid. On August 23rd there were 8,000 cells; on August 27th nearly 26,000 cells; on September 4th, 8,000 cells again; September 5th, 63,000 cells, and the last puncture, on September 18th, showed over 1,000 red blood cells in the spinal fluid. There was never any evidence of block in the subarachnoid space. On August 17th the spinal fluid pressure reached 630, and from August 21st on remained over 680, except on isolated occasions. The last lumbar puncture showed a spinal fluid pressure of 630. On August 22nd the patient complained of diplopia and there was a right 6th cranial nerve weakness. On that day also he became maniacal and irrational, and this remained more or less present to the end, with some relief after each lumbar puncture. Bilateral papillœdema increased gradually, with hæmorrhages into the retina, but no other localizing neurological signs. From August 23rd on, the upper and lower abdominal reflexes were absent; the neck was rigid; and there was a bilateral Kernig sign which remained up to the end. In addition to this, on August 27th there was albumin in the urine, with a large number of pus cells but with no blood or casts. On August 29th an increasing amount of albumin was present with hyaline casts and a large number of pus cells. The casts disappeared from the urine on August 31st as well as the albumin, and the latter returned from then on only in traces. Investigation of the genito-urinary tract by the genito-urinary specialists did not reveal any local cause.

On August 28th the temperature rose to 104.4° F., and examination of the chest revealed patchy areas of dullness in the right middle and lower lobes which, by x-ray, were reported due to partial atelectasis of the right lung. Although the physical signs of this condition varied from time to time, the atelectasis, by x-ray, remained.

In the meantime, in addition to the red blood cells in the spinal fluid, an increasing number of white cells were appearing, many of them large mononucleated cells. A large amount of fluid was collected, and to it was added an equal quantity of 94 per cent alcohol. After standing several hours the fluid was centrifuged, and the sediment dehydrated, imbedded in paraffin and sectioned. It was then seen that many of the large cells were undergoing mitosis (Fig. 1). They were about three to four times the size of a leucocyte and were definitely considered to be tumour cells. Similar cells were seen in the urine but there their exact nature was less certain. On September 16th the patient developed a dysarthria and a weakness of the left face of the peripheral neuron type.

On September 17th bilateral arterial encephalography with thorotrast was done by Dr. Arthur Elvidge. Our interpretations of the films were inconclusive.* On September 19th he developed urinary retention and had to be catheterized. The left facial weakness was complete, upper and lower; there was weakness of the left arm and leg, and there appeared to be some anæsthesia of the inner part of the left leg. Both ankle jerks were absent. The mental condition of the patient made accurate examination impossible. He was incoherent, irrational, and had visual hallucinations. The peripheral signs, however, pointed to the presence of a cord tumour. Repeated examination revealed no cranial bruit. The patient gradually became worse and died on September 22, 1936.

At autopsy the following was found. There was hypostatic congestion and collapse of the right lung with extrapleural hæmorrhages. On the left side there was a bronchopneumonia. Sections through the lung failed to reveal any neoplasm. Cloudy swelling was present throughout the kidneys and liver. A tumour was found on the spinal cord at the level of the tenth dorsal vertebra and another in the cauda equina.

On examination of the brain there was no evidence of tumour on inspection or palpation. The dura was not tense, and there was only a small amount of blood in the subarachnoid space along vessels over the frontal pole and the tip of the right temporal pole. There was no flattening of the convolutions or undue widening of the sulci, but the arachnoid around the base was thickened and partially opaque. Dissection of the vessels revealed no abnormality.

On sectioning the brain† there was no asymmetry of the cerebral hemisphere and no ventricular shift or dilatation except for an enlarged left posterior horn. At the junction of the body of the right lateral ventricle with the temporal horn a reddish-brown tumour was found, about 3 cm. in diameter. At the level of the foramen of Munro and above this the right occipital horn was filled with tumour, which invaded the corpus callosum posteriorly (Fig. 2). The tumour was soft, fleshy, reddish-brown, with occasional greyish-yellow areas; there was some blood clot attached to it and hæmorrhage within it. In both the anterior horns there were many hæmorrhagic areas and apparent tumour implants on the ventricular walls. Similar implants were found on the medial wall of the body of the right lateral ventricle for a distance of 2.5 cm. anterior to the tumour mass. In one area the choroid plexus was united with the tumour, though, grossly, it was not clear whether this was due to adherence or whether the tumour was an outgrowth from the plexus.

At about the level of the tenth dorsal vertebra, on the surface of the spinal cord, a small rounded firm area was palpable. The tumour metastasis in the cauda equina consisted of a mass of reddish-brown tissue, semi-friable, very loosely adherent to the roots but not to the meninges otherwise, and with a very thin capsule, if any.

Microscopic sections of the cerebral neoplasm, made through many areas, showed a highly cellular tumour, diffusely infiltrating the surrounding cerebral tissue. There was tremendous variation in size and shape of the tumour cells, from approximately 15 to 150μ in

* These films were seen later by Dr. J. Sanchez-Perez, of Madrid, and he was able to localize the neoplasm from them.

† For the following pathological descriptions we are indebted to Dr. William Cone, Pathologist of the Montreal Neurological Institute, whose reports we have in part freely abstracted.

diameter. Many of the smaller cells were oval, others round. The huge ones were irregular, multinucleated giant cells, and some of them presented a multilobulated appearance.

The arrangement of the tumour cells followed no definite pattern and varied from area to area. In some parts they were found in long bands, and here the cells followed a definite polar arrangement like spongioblastoma polare, yet everywhere in such areas there was a tendency to form giant neuroglial cells, as in glioblastoma multiforme. In some areas the cells were in small islands, and in others they lined up both perpendicularly and concentrically about blood vessels. Phosphotungstic acid-hæmatoxylin stains revealed that many of the oval tumour cells had long tails. Metallic stains showed a great number of glial fibres and demonstrated many heavy glial bands in the tumour area, with a concentric arrangement around blood vessels and a great deal of clasmatodendrosis. There were many areas of focal degeneration. The pons and medulla were diffusely invaded, and sections through the right anterior horn showed tumour beneath the ependyma in the perivascular spaces and also scattered diffusely in the cerebral tissue near the ventricle.

One of the most significant findings was the tremendous extent of tumour diffusion through the meninges and choroid plexuses. In addition to the usual vascular changes that one sees in glioblastoma multiforme, consisting of endothelial and adventitial proliferation within the tumour and in the adjacent cerebral tissue, there was a very wide infiltration of tumour cells of glial origin around meningeal and choroid plexus vessels in the neighbourhood, and even remotely. These extensions consisted of thick perivascular collars of tumour cells (Figs. 3 and 4) which in some areas were so dense that they completely obliterated the normal adventitia of the vascular wall. In many cases tumour cells had penetrated and destroyed the muscular lining and reached down to the endothelium, eroding through it in some places. This undoubtedly must have been the cause of the numerous repeated massive subarachnoid hæmorrhages.

The thickened area at the level of the tenth dorsal segment of the spinal cord was composed of tumour cells, and similarly the mass of tissue about the cauda equina was found to be made up almost entirely of tumour cells. In the choroid plexus, in the meninges, and in the metastatic tumour cell groups the cells were rounded and similar to those isolated from the spinal fluid ante mortem.

Because of the tremendous cellular variability, the areas of necrosis, the vascular changes, and the wide diffusion, as well as the extremely rapid course, a diagnosis was made of glioblastoma multiforme involving the right occipital lobe, the splenium of the corpus callosum, with metastasis into the ventricles, spinal cord and cauda equina.

The following case bears a striking resemblance to Case 1, but ran a much shorter clinical course.

CASE 2

H.S., male, aged 42, was referred to the Montreal Neurological Institute on May 15th, 1936, by Dr. L. Notkin, of Montreal, with the following history.

He was perfectly well until five weeks before admission, when he developed a cold in the head which lasted about three weeks. With the disappearance of this cold he began to complain of a dull, aching, steady pain in the occipital region of his head on both sides, radiating towards and soon involving the frontal region bilaterally. This had been constantly present for two weeks prior to admission, and for the last ten days he had felt drowsy yet unable to sleep because of headache. On one or two occasions there had been slight fever. He had vomited three or four times after meals, and there had also been transitory photophobia.

The family and past personal histories were irrelevant. On admission, the patient was drowsy, resentful and uncooperative. He complained bitterly of dull aching generalized headaches. The ears, nose and throat were not remarkable. The chest and cardiovascular system were normal; blood pressure, 136/76; and the prostate was normal. There was photophobia. The margins of both optic discs were blurred and indistinct, especially on the nasal side, giving the impression of an early papillœdema. There was generalized hyperreflexia, but the superficial reflexes were intact and the neurological examination was otherwise essentially normal.

Laboratory data.—Urine: on admission, normal. *Blood count.*—24,000 white blood cells, with relative polymorphonuclear leucocytosis.

Lumbar puncture.—On admission showed an initial pressure of 300 mm. of water; a faintly yellow fluid containing 360 red blood cells and 60 white blood cells per c.mm. (mainly lymphocytes); a positive Pandy test and 63 mg. per cent total proteins.

X-rays of skull.—Antero-posterior and stereo left lateral views showed a somewhat decalcified dorsum sellæ, with calcification posterior to it and probably in the petro-clinoid ligaments. The pineal body was not calcified. The right antrum was denser than normal. According to Dr. Arthur Childe, roentgenologist of the Institute, the appearance of the skull was compatible with early increased intracranial pressure.

X-ray of chest.—Was essentially normal.

In view of these findings the tentative diagnosis of a recovering subarachnoid hæmorrhage was made. At the same time the insidious onset of the headache and other findings suggested the possibility of a neoplasm. Lumbar punctures were repeated to relieve headache, and on May 18th the initial pressure was 175 mm. H_2O, and the fluid was still faintly yellow and contained 30 red blood cells and 112 white blood cells with a faintly positive Pandy test and 52 mg. per cent proteins. On May 26th the pressure was 250; the spinal fluid was very faintly yellow and contained 64 white blood cells per c.mm. Many of these cells were mononucleated and larger than ordinary lymphocytes. They were suspected of being neoplastic, but after fixing and centrifuging the sediment and preparing paraffin sections no definite tumour cells could be identified.

Blood and spinal fluid Wassermann tests were negative. Irrigation of the right antrum showed a slight amount of shreddy material but no pus. Ophthalmological consultation (Dr. Tooke), May 19th, confirmed the slight degree of blurring of the upper margins of both nerve heads, and in the right eye there was a small amount of lymph exudate.

The patient remained difficult to nurse, persistently demanding attention; he showed a marked lack of insight, was unreasonable and very restless. On one occasion he insisted that he would leave the hospital and was persuaded to stay only after a great deal of difficulty. On May 27th the papillœdema showed a definite advancement. There was a quick nystagmus on gaze to the left and slow nystagmus on gaze to the right. A slight droop of the left upper lid was present but the cranial nerves were otherwise normal. There was a very slight uncertainty in finger-nose-finger test and heel-knee-tibia test on the left side, as compared with the right. The left upper abdominal was easily fatigued and position-sense in the left fingers and toes was diminished. Like Case 1 he was constantly complaining, demanding, and easily distractible. In this case also visual fields could not be plotted because of lack of attention. Visualization of the ventricular system was considered, but delayed because of the menace of re-exciting a subarachnoid hæmorrhage. The following day he became more confused mentally, and on May 29th at 12.30 a.m.,

the patient suddenly became comatose, developed Cheynes-Stokes respirations, and died.

At autopsy the brain showed a generalized pallor and flattening of the convolutions, with some softening at the surface of the right occipital lobe. There was little blood in the basal cistern and none in the subarachnoid space. A well marked pressure cone was present around the cerebellum and medulla. Sections of the brain (Fig. 5) revealed, grossly, a large neoplasm in the right occipital lobe completely obliterating the right posterior horn. It measured 7 x 4 cm. and displaced the ventricular system to the left. The tumour invaded the posterior end of the basal ganglia and the internal capsule, and extended into the posterior part of the corpus callosum. Antero-medially a firm piece of tumour impinged on the third ventricle, and just external to this was a spongy hæmorrhagic mass. Posteriorly there was a large cyst (4 x 5 x 2 cm.) within the tumour, filled with a faintly greenish gelatinous substance. Microscopically, sections of the tumour showed the typical wide variability in kinds of tumour cells characteristic of glioblastoma multiforme. There were many areas of hæmorrhage, necrosis and endothelial proliferation. The large cyst was rather an unusual feature for this type of tumour.

DISCUSSION

Because of their similarity it is convenient to consider these two cases before presenting the third. In summary, both gave a history sugges-

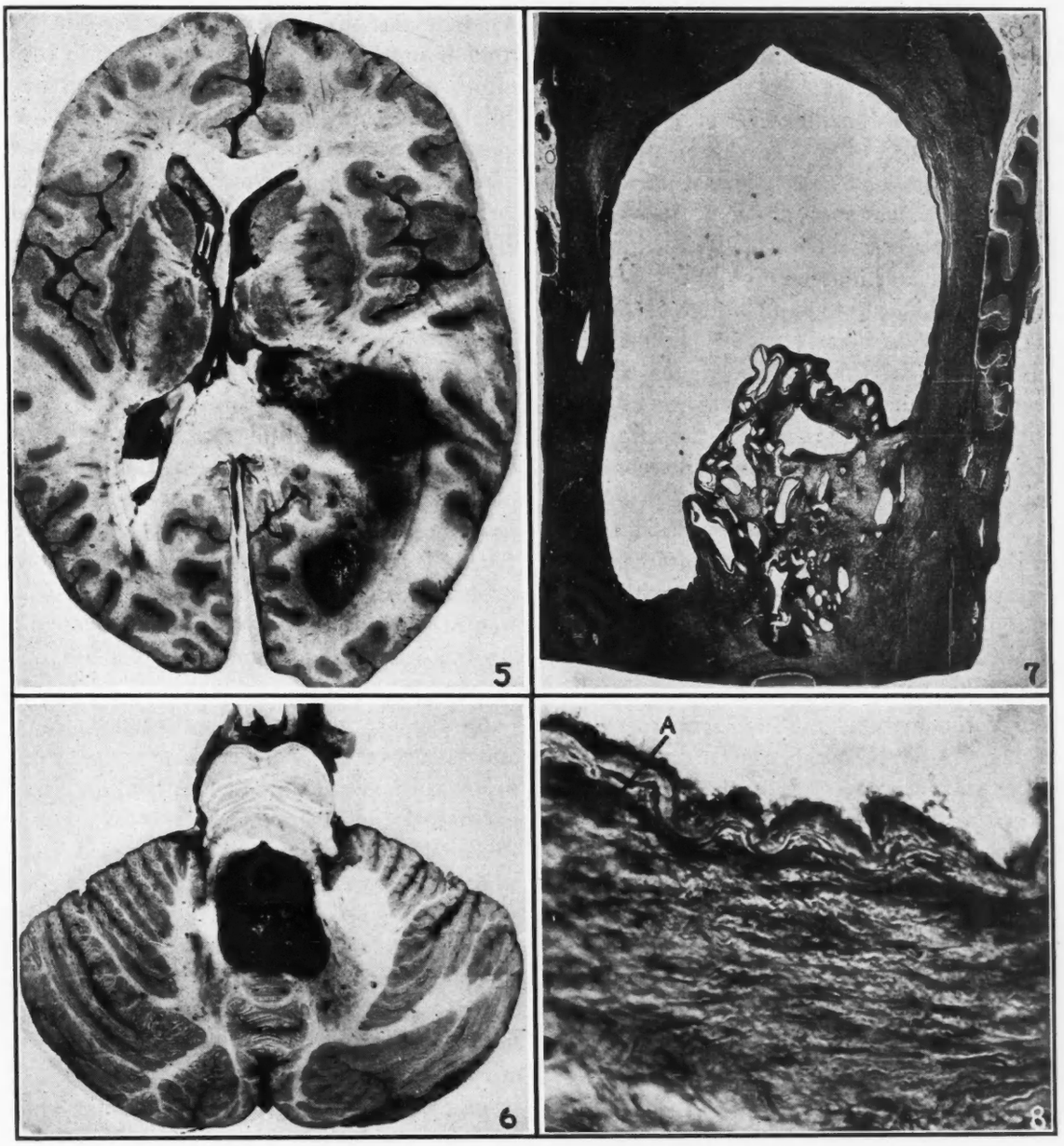

Fig. 5.—Case 2. Horizontal section of the brain showing tumour in the right occipital pole and involving the splenium of the corpus callosum. A cyst is present in the posterior part of the tumour. **Fig. 6.**—Case 3. Horizontal section through the cerebellum showing tumour and blood clot occupying the fourth ventricle. **Fig. 7.**—Case 3. Section through the fourth ventricle showing the cyst-like tumour composed of blood vessels. **Fig. 8.**—Case 3. Higher magnification of one of the blood vessels comprising the lesion. At "A" there can be seen an incomplete elastica interna.

tive of spontaneous subarachnoid hæmorrhage. On admission there was a striking absence of localizing signs and symptoms. By lumbar puncture blood was found in the spinal fluid, and no evidence could be elicited to warrant any diagnosis other than subarachnoid hæmorrhage. In the first case there were many repeated attacks of the typical syndrome. In both of them at autopsy a glioblastoma multiforme was found involving the right occipital pole and the splenium of the corpus callosum, with a varying amount of local and general metastasis in the brain and spinal cord.

The cause of the subarachnoid hæmorrhage in each case is quite clear. In the first there was extension of the tumour cells in perivascular collars along large vessels in the meninges and choroid plexuses. These perivascular collars became dense masses of growing tumour cells which in many cases obliterated and destroyed the adventitia and muscular coats of the vessel walls, penetrating up to and through the intimal lining, with subsequent massive hæmorrhage. Various stages of this erosion would be sufficient to maintain a constant seepage of blood through damaged vessels and so produce blood in the cerebrospinal fluid. In the second the appearance of blood in the spinal fluid must be laid to the proximity of the neoplasm to the lateral and third ventricles, the necrosis and degeneration with vascular changes within the neoplasm and subsequent hæmorrhage. Death probably occurred from sudden hæmorrhage into the third ventricle.

One of the features in Case 1 is worth emphasizing. A large number of neoplastic and mitosing cells were found in the spinal fluid. Exactly similar cells were found in the tumour mass, in its advancing borders, and seeded through the meninges. Examination of the spinal fluid by the method outlined should be done as a valuable diagnostic measure in all obscure cases of subarachnoid hæmorrhage.

The leucocytosis which was present in both these cases is commonly found in spontaneous subarachnoid hæmorrhage, and no satisfactory explanation can be offered for it. According to Wilson,[13] however, leucocytosis and neutrophilia occur in about 33 per cent of cases of glioma.

As far as albuminuria is concerned, this has been a common finding in subarachnoid hæmorrhage. It was first recorded by Widal in 1903, and various causes have been assigned for it. Widal felt that it might be caused by lesions of the brain stem, and in Claude Bernard's classical work on experimental lesions on the floor of the fourth ventricle he mentions albuminuria as one of the findings often associated with glycosuria. Clinically, it has been thought that the albuminuria might be an evidence of direct irritation of the kidneys as a result of sudden changes in systemic pressure, following on the sudden increase in intracranial pressure due to the subarachnoid hæmorrhage. In this particular case the blood pressure was recorded every four hours during the patient's stay in the hospital, and despite the very wide variations in his intracerebral pressure, which at each lumbar puncture was reduced from 600 or more to zero, no corresponding change was found in his blood pressure nor were there any sudden changes in blood pressure immediately before his hæmorrhagic episodes. This, in retrospect, should have suggested that the blood in the spinal fluid was caused by vascular degeneration within a neoplasm rather than a sudden vascular accident such as the bursting of an aneurysm, etc. In the latter case definite changes in the systemic blood pressure are usually found before and after the subarachnoid hæmorrhage. The tumour was found to infiltrate the floor of the fourth ventricle very extensively, and it seems probable that the irritation consequent on this was responsible for the albumin and casts that showered into the urine at one phase of the disease.

As far as the question of earlier diagnosis and treatment in these cases is concerned, the problem of visualizing the ventricles arose frequently for discussion. It was thought that encephalography should be postponed till the spinal fluid was clear. The delay we felt was justified because of the dangers of stirring up another hæmorrhage by introducing a foreign substance that might irritate the bleeding point, dislodge the clot, and start hæmorrhage again. As it happens, in Case 1 the procedure would not have been helpful, because there was, even at autopsy, no shift of the ventricular system. In the second case we apparently waited too long. In retrospect the most important single factor is the history. True spontaneous subarachnoid hæmorrhage caused by aneurysm or arteriosclerosis is initiated with hammer-like

suddenness by headache. In both these cases the symptom of headache was gradual in onset. The question of the advisability of visualizing the ventricles must rest on the individual case.

Several points not directly concerned with the question of subarachnoid hæmorrhage are, however, worth mentioning. In both of these patients similar mental reactions were found, consisting of lack of insight, inattentiveness and unreasonableness. Analysis of these symptoms suggests that agnosia was also present. In neither case was there any involvement of the frontal poles except for slight subependymal implantations in one of them. The neoplasms involved the occipital pole and the splenium of the corpus callosum. The former can be ruled out as responsible, and one can also dismiss the question of increased intracranial pressure. It seems fair to ascribe these mental changes to involvement of the splenium of the corpus callosum. This is in agreement with the observations of a number of other authors including Alpers and Grant,[14] Alpers,[15] Cramer,[16] Voris and Adson.[17]

The following patient is one of the group which was previously reported,[2] but the sequel can now be written.

CASE 3

L.V., female, a patient of Dr. H. S. Shaw, was first admitted to the Royal Victoria Hospital on December 12, 1931, at the age of 13. One month before, she had had a mild attack of grippe from which she recovered perfectly well and she returned to school. Five days before she had developed a slight fever, but this disappeared the following day. The day before admission, on arising, she had had a slight headache, and shortly afterwards developed a severe pain in the back of the neck, which was accentuated by bending her head forward. Vomiting began soon after the pain and both these things, accompanied by increasing restlessness and irritability, persisted.

There was no history of trauma, and her family history and functional inquiry were essentially normal. On examination she had a stiff neck and a bilaterally positive Kernig sign; the deep reflexes were slightly hyperactive on the left side, but the superficial reflexes were normal. The patient was very irritable, and movements of the head caused a great deal of pain. There was a suggestion of facial asymmetry about the mouth, but both sides moved equally well. The tonsils were large and infected, but the heart and lungs were normal. Blood pressure was 128/80. The urine and blood counts were normal; the blood Wassermann test was negative. X-rays of skull and chest were normal. The visual fields were normal. No cranial bruit was audible.

Lumbar puncture, on admission, showed an initial pressure of 410 mm. of water. The fluid was grossly bloody, and contained 215,000 red blood cells per c.mm. The stiffness of the neck, positive Kernig sign and reflex changes disappeared in 4 to 5 days, and the blood in the cerebrospinal fluid gradually disappeared. Further investigation failed to reveal the cause of the hæmorrhage, and the patient made an uneventful recovery.

She remained perfectly well until five months later, when she noticed a slight headache and vomited two or three times. There was slight stiffness of the neck, which disappeared the following day. Nine days later, on arising in the morning, she complained of headache and gradually increasing rigidity of the neck accompanied by persistent vomiting. Examination again showed stiffness of the neck and a bilaterally positive Kernig sign. There was again a slight questionable weakness of the left lower face, due more to an asymmetry than real muscular weakness. The knee-jerk on the left side was hyperactive, and the left abdominal fatigued more quickly than the right. Blood pressure was 114/86. Physical examination was otherwise normal. A lumbar puncture was done, which showed an initial pressure of 180 mm. of water and grossly bloody fluid containing 56,400 cells. The blood and urine were again normal, and the blood Wassermann test was negative. The patient made an uneventful recovery and left the hospital in 20 days—the physical and neurological examinations being normal.

Nine days after discharge, on August 26, 1932, there was a recurrence of the previous clinical syndrome, and again 375,000 red blood cells were found in the cerebrospinal fluid. The fluid gradually cleared. While in the hospital, on September 6, 1932, the patient had a spell starting with numbness in the fingers of both hands, which extended up to the wrists. Then both hands and feet became stiff and her arms flexed at the elbows, with her wrists and fingers extended. This was followed by an exacerbation of the rigidity of the neck. This attack was followed by severe frontal headache. Examination immediately after showed the blood pressure, 100/60; pulse, 80; respirations, 22; and temperature, 97.2° F. The pupils were equal and active, and both knee jerks were increased. The right ankle jerk was increased as compared with the left and had a transient clonus; both abdominals were equal and active, but the right plantar response was questionable. Lumbar puncture showed a marked increase in the number of red blood cells in her spinal fluid. Following this episode again the patient recovered and by October, 1932, her neurological examination was essentially normal. A tonsillectomy was done and she was discharged.

The next attack of headache and stiffness occurred two months later, and she was again admitted to the hospital, where it was found that another subarachnoid hæmorrhage had occurred. There were no localizable neurological signs. Following this the patient remained well for about two months, except for an occasional headache, until the beginning of September, 1934, when there was a recurrence of stiffness of the neck and vomiting. This time there was persistent right ankle clonus and transient left ankle and patellar clonus, with weak abdominal reflexes on the right side. Again her cerebrospinal fluid was bloody. Despite this, on repeated determinations, the cerebrospinal fluid proteins were within normal limits.

The next hæmorrhage occurred ten months later on October 11, 1935, and on this occasion there was found an early papillœdema in the right eye; the knee and ankle jerks were slightly more active on the right than the left side, and only the left lower abdominal reflex could be obtained. There were about 250,000 red blood cells in the cerebrospinal fluid. This time, when the hæmorrhage had subsided, bilateral arterial encephalography was carried out by Dr. Elvidge. The arterial systems of both hemispheres were well outlined and no definite abnormality was shown. No aneurysm was demonstrated. Ventriculography was also done by Dr. Elvidge, and symmetrical moderate dilatation of the ventricular system was found.

The papillœdema, which had soon involved both eyes, subsided, and by November 12, 1935, her lumbar puncture was again normal, the fluid was clear, and neurological examination normal. Again, throughout

this episode the cerebrospinal fluid proteins remained within normal limits. On November 16th the patient had three attacks of dizziness, during which objects moved in a clockwise direction. These attacks lasted several minutes, and on examination there was a nystagmus on gaze right, left and upward, perhaps slightly more marked on gaze to the right. The following day there was a very slight ataxia of the right hand and some also in the left hand. This disappeared in a day or two.

The final attack occurred on February 19th, 1936, when the patient complained of feeling dizzy on arising. Ten minutes later she said she was unable to hear anything, and soon after she lost consciousness. On admission she was unconscious, very pale, and her blood pressure was 140/96. Her pupils were small and equal in size and responded to light. There was bilateral skew deviation, and slight spontaneous nystagmus with slow phase to the left. Her limbs were flaccid. The deep reflexes in the lower limbs were increased; the abdominal reflexes were absent, and there was bilateral plantar extension. Her cerebrospinal pressure was 220 mm. of water, and the fluid was very bloody, (175,000 red blood cells per c.mm.). Her condition gradually became worse, and spasmodic jerks of the arms and legs developed. Several hours later her legs became spastic, respirations remained regular but shallow, and she became cyanosed. Her condition gradually became worse and she died.

In summary.—This girl had the first attack of subarachnoid hæmorrhage at the age of 13. From then on till the time of death, at the age of 18, she had nine episodes consisting of sudden onset of severe pain at the back of the neck accompanied by headache and persistent vomiting. With each episode she had stiffness of the neck, a bilaterally positive Kernig sign and a large amount of blood in the spinal fluid. At first there were transient signs of a slight left pyramidal lesion; in later attacks the signs pointed to a right pyramidal lesion, but all signs cleared when the acute period subsided. On several occasions a slight and indefinite weakness of the left lower face was noted. One hæmorrhage was initiated with a convulsive seizure involving both arms and legs at the same time. General physical examination revealed no possible etiological factor. Arteriography and ventriculography were equally non-productive in providing a clue to the cause of the hæmorrhage. Following each episode the neurological examination became essentially negative, and over a period of 8 to 15 days the spinal fluid pressure and cells became normal. Even immediately after each hæmorrhage the spinal fluid proteins remained within normal limits, despite the enormous amount of bleeding—a phenomenon for which no explanation can be offered. On one occasion the patient had an attack which seemed to be cerebellar in origin and was followed by slight cerebellar signs. At no time was any cranial bruit audible. With the last hæmorrhage there was skew deviation of the eyes and a slow spontaneous nystagmus to the left.

At autopsy.—Examination of the brain showed that the subarachnoid cisterns at the base were filled with quite bloody fluid. The floor of the third ventricle was tense and bulging, and the arachnoid over the cisterns was thick and tough. The circle of Willis was dissected and its tributaries and branches examined but no aneurysm or bleeding point was seen. There was considerable blood around the foramina of Luschka and Magendie and in the fourth ventricle. A marked pressure cone was present about the tonsils of the cerebellum, and considerable thickening with bile-stained pigmentation of the leptomeninges of the cisterna ambiens.

On sectioning the brain, a symmetrical enlargement of the lateral ventricles, the third ventricle and the aqueduct was found, the latter being 0.4 cm. in diameter. The superior part of the lateral ventricles, third ventricle, aqueduct, and dilated fourth ventricle were filled with blood clot (Fig. 6).

Arising from the roof of the fourth ventricle and extending down into it was a spade-shaped structure, measuring 1.4 x 1.2 x 1.1 cm. This appeared to be a mass of blood vessels and connective tissue. There were numerous cavities in this mass, suggesting small cysts (Fig. 7). Microscopically it was found that protruding into the cavity of the fourth ventricle from its roof was a mass of blood vessels lying within glial tissue. These vessels varied in size and all of them had thin walls. Many of them had a partial elastica interna which was incomplete (Fig. 8). Other vessels did not show this structure and were apparently veins. There was one area of recent hæmorrhage deep in the glial tissue near the overlying cerebellum. No tumour cells were seen in any area, nor structures suggesting a true proliferative neoplasm. There were many chronic inflammatory cells and phagocytes containing iron pigment around this mass and in the leptomeninges.

A pathological diagnosis of arteriovenous angioma was made.

DISCUSSION

In their classical monograph on blood vessel tumours of the brain (1928) Bailey and Cushing distinguished clearly between hæmangiomata and angiomatous malformations. The former are definitely neoplastic infiltrative lesions, and to this group belong Lindau's and von Hippel's disease, etc. The angiomatous malformations, on the other hand, may be (*a*) simple telangiectases or capillary loops; (*b*) venous angiomas—simple, serpentine or racemose (cirsoid) made up of venous channels, and (*c*) arterial or arteriovenous angiomas (angioma arteriale). They consist of misshapen vascular loops which are essentially embryological anomalies, and even though they are capable of growth they are not infiltrative tumours like hæmangiomata. The arteriovenous type of angioma may be aneurysmal in the sense that the vessels dilate and may contain a direct shunt in which blood flows directly from the artery supplying them into the venous channels, and Bailey and Cushing refer to them as aneurysmal angiomas. For the same reason certain authors have described them simply as arteriovenous aneurysms, and so created a certain amount of confusion in their nomenclature in the literature. Thus Dandy[18] in his monograph refers to these lesions as arteriovenous aneurysms, and Bergstrand, Olivecrona and Tönnis[10] recently have preferred to retain this term. Confusion arises in distinguishing between lesions of this sort and the simple saccular expansions to which the term aneurysm is generally applied. Such aneurysms are caused by the ballooning of the wall of an artery due to a defect in its wall,

and of course are found very commonly in the arteries of the circle of Willis. Such a lesion is obviously different from a cluster of improperly formed vascular channels, and the term angioma would seem to be more satisfactory for the latter.

Many of the vascular channels in our case had an imperfect elastica interna, and it seems clear that we are dealing with a true angioma arteriale of Bailey and Cushing. No other angiomatous lesions were present in the body. The repeated attacks of subarachnoid hæmorrhage were doubtless due to deficiencies and premature degenerative changes in the vascular channels comprising the lesion.

As a matter of interest it is noteworthy that this particular location in the roof of the 4th ventricle is a very rare one for arteriovenous angiomata. According to Lindau[19] they occur almost exclusively above the tentorium cerebelli, in contrast to hæmangioblastomata which are always cerebellar in location. However, one case was recorded in Bailey and Cushing's series occurring in the left cerebellar hemisphere (Case 10), and they collected five others reported in the literature. Dandy[18] described one case, and another was operated on by Olivecrona and Lysholm.[20] The latter case resembles our patient to some extent in that it occurred in a boy aged 16 who for 1½ years had been complaining of attacks of headache and vomiting, which were probably due to subarachnoid hæmorrhage. However, when he came to the clinic he presented definite cerebellar signs as well as cranial nerve and motor weaknesses. At autopsy a large midline cerebellar tumour was found obliterating the 4th ventricle. The tissue was studied histologically by Bailey and Cushing, and classified as an angioma arteriale. Two cases reported by Tönnis were in the posterior fossa, and one of these (No. 15) was the first recorded example of a successful extirpation.

SUMMARY AND CONCLUSIONS

1. The occurrence of the syndrome of subarachnoid hæmorrhage during the pre-operative course of brain tumours is extremely rare in the literature; only 1 example of glioma and 8 cases of blood vessel tumours have been previously reported.

2. Two cases of glioblastoma multiforme and 1 case of arteriovenous angioma which gave rise to this syndrome are presented.

3. Both these glioblastomata occurred in the right occipital pole and involved the splenium of the corpus callosum, and it is felt that the latter gave rise to distinctive mental changes in these patients.

4. Tumour cells containing mitotic figures were found in the spinal fluid in one of these glioblastomata. The tumour in this case showed an unusually wide metastatic spread.

5. Brain tumour must be considered in the differential diagnosis of spontaneous subarachnoid hæmorrhage.

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PRACTICAL PERIMETRY: CONSTRUCTION AND OPERATION OF THE TANGENT SCREEN*

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THE findings obtained by perimetry are of first importance in many ophthalmological conditions, and also have become practically indispensable in neurological organic diagnosis. Due to the forward swing of the optic radiation around the tip of the temporal horn of the lateral ventricle, the visual channels extend nearly twice the length of the head, and are in intimate relation to many structures en route which are capable of altering the function of these pathways. Indeed, there are few pathological processes occurring within the orbital or cranial cavities[9] which do not at one time or another interfere with the function of these sensory routes.

I have journeyed today not as an apostle of something new, but only to plead for more widespread use of the tangent screen, and to present in homely terms a practical and inexpensive device for obtaining visual fields accurately and with casual easiness, when surrounded with a few safeguards. The ponderous glittering apparatus of manufacturers, and the earnest refinements of research workers have seemed in recent years to introduce such bewildering nicety into the art of perimetry that its clinical utilization is diminishing through timorousness and through awe of a few unessential geometric seconds of error. Meanwhile, here in the Pacific northwest, lesion after lesion — obvious with perimetry—is being regarded as obscure, and diagnosis is uncertain or frankly missed because of lack of its employment.† Routine quantitative perimetry[10] can be done in 15 to 20 minutes without fatigue to the most uncooperative patient or to the examiner; Bjerrum, fifty years ago, used merely the back of his consultation room door.[4] It is concerning this casual and accurate method that I wish to speak.

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† The purpose of this paper, frankly, is to answer the eager, "How is it made? How is it used? What is found?" The equally important "Why are the findings there?" is merely touched upon in closing.

CONSTRUCTION

Screen.—A perimetric screen is laid out on trigonometric principles and may be any size or shape, but since planning in terms of degrees, sines, and tangents often appears confusing—simple though it actually is—the detailed construction given below is in terms of feet and centimetres. For the tangent screen, the junction of each thirty degrees of circumferential arc and each ten-degree interval outward from the fixation point are marked upon a stretched black cloth. The accuracy and utility of a tangent screen diminishes rapidly beyond 35 to 38° from the fixation point, so that it is not customary to have the screen more than 70 to 80° wide, but inasmuch as probably 95 per cent of all significant ophthalmo-neurological lesions are quantitatively discoverable readily within this area, supplemental use of the fussy and exhausting 180° perimeter is a rare necessity.

In planning a screen the first thing to determine is the distance which available space will allow one to place between the patient and the fixation point on the screen; this is usually 1,000 mm., 1,760 mm.,* or 2,000 mm.;[11] in general, the larger the screen, the easier and more accurate is the exploration. If one will next lay out the determined distance to scale on a piece of paper; represent the screen by a line drawn perpendicular to this; represent the true visual field by a semicircle whose circumference passes through the fixation point, with the patient as its centre; mark each ten degrees upon this circle by means of a protractor, using the fixation point as zero; extend the lines from the centre of the circle through the marked points, so as to intercept the represented screen; one may then read to scale (by aliquot parts) the radial distances outward from the fixation point which each of the ten-degree markings should occupy on the tangent screen. The following description is for the recommended two-meter fixation distance.

A frame of one-inch iron pipe is made, 8½ feet square, with a central upright brace across it (Fig. 1); over this is laced a stretched covering of black canton flannel† provided with tent flanges at the lacing holes. The frame is then tied upright to the picture-moulding of the examining room by 2 flexible loops of ordinary woven picture-wire. A circular patch of adhesive tape 2 cm. in diameter, stained pale grey with dilute ink, is placed in the centre of the screen (fixation disc) 51 inches above the floor. From this is stretched successively a piece of chalked string, so as to mark radii dividing the screen into four 90°

* This unusual distance is sometimes chosen because it corresponds to the occasionally-used 6-foot distance for testing acuity. In practice, 2,000 mm. (2 metres) is standard, and normal isopters then fall in nearly substantial agreement with standard chart markings; actually, 1/2,000 is found to be about 11 to 12°; 2/2,000 about 21 to 25°, etc. The brilliancy of test objects and of illumination affect these absolute values considerably, but the relative values and defects found to be useful clinically are reasonably independent of such niceties, since form of field and relative extent are of principal importance.

† Velvet and seersucker have a distracting sheen; felt soon sags badly.

quadrants; each quadrant is then similarly divided into thirds. On each of the twelve chalked radii other small cross-marks are made 35 cm., 72 cm., and 114 cm. from the fixation point; these junctions are then marked permanently by a small tuft of black yarn sewn into the screen (Fig. 3); the chalk marks are now brushed off. The blind spot, 17 cm. by 26 cm., is marked bilaterally with 4 tufts of yarn, as shown, the inner border being 43 cm. from the fixation point.

5 mm., 7 mm., 10 mm., etc., mounted on black pins* are severally inserted in the end of a black 12-inch wand (gynæcological applicator), to be held in the perimetrist's hand and moved inward along the radii of the screen (see Fig. 3).

The results are recorded as minute circles on the perimetric chart, and these points connected by straight lines (see Charts); the Charts are 8½ x 11 inches, large enough to avoid crowding of lines, and are printed in

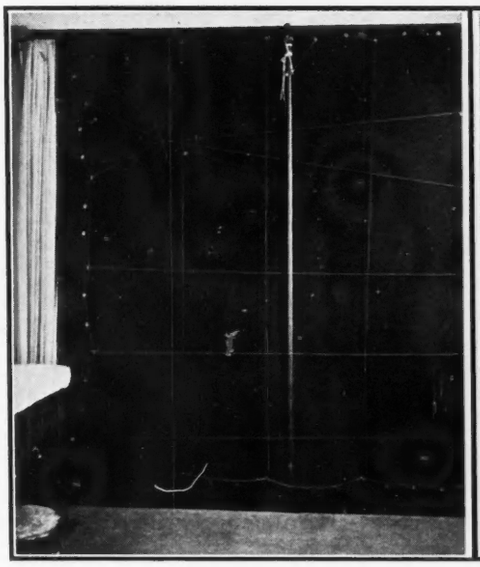


Fig. 1

Fig. 2

Fig. 1.—The back of the tangent screen, showing the central brace and the manner in which the black cloth is stretched by lacing through reinforced holes.
Fig. 2.—Shows the front of the screen in operation.

If the large black screen makes the room appear dull for ordinary use, a pongee draw-curtain on automatic rails may be suspended before it when it is not in use.*

Lighting.—In order to assure uniform conditions, so that serial results are comparable, artificial lighting is necessary. The room is darkened with opaque black window shades (which need not be completely light-proof), and the screen is illuminated by two 750-watt lamps in ventilated reflectors, fronted with milk glass, placed high in the corners of the room just behind the patient. Blinding illumination is not necessary; visual acuity increases practically not at all beyond 8 to 9 footcandles of illumination,† and nothing is added (except complication and expense) in having more.

Accessories.—To avoid distracting the patient's attention from the small test objects, the perimetrist dons a long black surgical gown, into the sleeves of which have been sewn black cotton gloves (Fig. 2); a hood is unnecessary. The patient is seated in a comfortable arm-chair, so placed that his head is closely approximate to 2 metres distant from the fixation disc. One eye is covered by a comfortable aluminum or brass eyecup painted black, held lightly in place by a clip of spring wire passing over the vertex to a small brass pressure plate at its other extremity over the occiput (Fig. 5). White cardboard discs, 1 mm., 2 mm., 3 mm., neutral orange or light maroon, so that the plotted fields (recorded in black ink) stand out brilliantly upon them.†

PROCEDURE

When the shades have been lowered, room lighted, screen disclosed, visual acuity (Snellen, and maybe Jaeger) recorded with and without glasses, gown donned, and patient seated comfortably in the chair facing the screen, the right eye is covered with the spring cup, and one says:

Here in the centre of the screen is a gray spot. I want you to watch it all the time. On the end of this stick I have a smaller white spot which will be moved about; you will not be able to see this little moving white spot all the time. I want you to say "Yes" just as soon as you see the little one. But you must *watch* only this central gray spot, and must not take your eye off it. You understand? . . . . The first couple of times don't count, so there's nothing very deadly about it;

* Pipe frame, $6; canton flannel, $12; pongee, $18; window shades, $8; flood lights, $20; gown, $3; eyecup clip, $2; test objects, $3; charts, $4 per hundred, in small quantities.

† If desired, this may be determined objectively with one of the (Weston) photoelectric cells now commonly used in professional and amateur photography.

* These Walker *pin discs* can easily be kept brilliant by a thick coating of water-colour Chinese White, and need be only moistened for an instant and wiped lightly to regain complete brilliancy. They are obtainable in sets from the United Superior Optical Company, 727 West 7th Street, Los Angeles, Calif.; if more elaborate *spherical objects* ($10) are desired, the Berens set may be obtained from E. B. Meyrowitz Surgical Instrument Company, 520 5th Avenue, New York City.

† The *perimetric charts,* with customary exaggeration of the peri-macular area (see Fig. 5, may be obtained from Neal Gulovson Co., 1516 N. E. 33rd Avenue, Portland, Oregon.

just watch the centre spot, and say "yes" when first you see something white and moving off here. All ready! Here goes.

One then moves the 2 mm. white disc inward along each radius (Fig. 2), and records mentally the spot at which the the patient says "yes", until all six meridans have been explored; then one says, "Close your eyes, so as to rest them, until I tell you again". One then goes to the recording desk, and, remembering the six meridianal "yes"-spots as, say, 22°, 22°, 22°, 23°, 25°, and 24°, marks these places on the appropriate meridans of the chart (Fig. 4); the other half of the eye is then explored, and recorded. (One may easily watch a poorly-co-operative patient's eyes, to be certain that fixation is maintained.) The eye cup is then changed to the opposite eye, and its fields are explored with the 2 mm. disc and recorded; then the 1 mm. disc is used on that same eye. Next, the eye cup is shifted, and, in the first eye, exploration is done with the 1 mm. disc and then with a 3 mm. disc; finally the right eye is also explored with a 3 mm. disc, and the size of the blind spot plotted, using the 3 mm. disc; the test is then completed by plotting the blind spot in the opposite eye. The whole procedure is done in an unhurried quiet manner, and need not take more than 12 to 20 minutes.

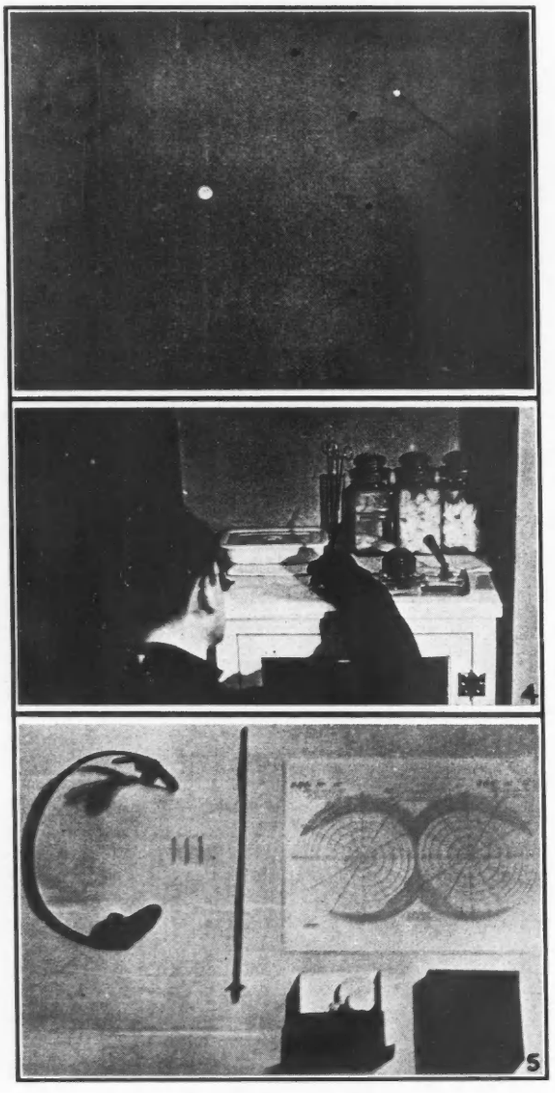


Fig. 3.—The circle of ten-degree markings of yarn about the fixation disc. **Fig. 4.**—Shows the manner in which the findings are posted in batches onto the chart; the top of an instrument cabinet is an excellent recording desk. **Fig. 5.**—The inexpensive small accessories mentioned in the text, used in tangent perimetry.

Comment.—This plan is far easier than exploration with small cramped perimeters, the patient sitting strainedly at a chin rest, and watching relatively immense test objects (customarily 10 or 15 mm. in diameter) moving inward; or with immense mechanical gadgets requiring as much care as a Diesel engine and equally distracting to the patient. Walker[10, 11] has shown that when *quantitative* perimetry is done with small white test objects such as these, exploration of *fields for colour* yields practically no additional information, and that even the smallest and vaguest scotomata may be mapped with relatively complete accuracy with 1 mm. or 0.5 mm. white discs as competently as with coloured objects.

The advantage of the above method is that it is simple, quiet, comfortable, invariable, and accurate; it may be used regularly as part of examination, with less fuss than even ophthalmoscopic study. Its importance is that it is workable, that it utilizes something which every trained person recognizes as important, but which at present is often thought of as being difficult, obscure, and almost too much bother to go through with. With such simple apparatus, and ready availability, valid excuse can scarce be advanced for omission of perimetry in any single necessary instance of examination.

Normal distortions.—If the patient slouches in his chair his eyebrows may prevent full delimitation of his fields upwardly; similarly, a large nose may encroach upon the lower nasal quadrants of his fields; or crossed knees may make a pseudo-indentation in his 3 mm. fields in the lower quadrants. Fields are plotted without glasses, and this at times may result in slightly ovoid fields, if heavy refractive error, or

more than a moderate degree of astigmatism, is present.

Pathological fields.—If a central scotoma precludes central fixation it is probably best to use a stereo-campimeter to plot fields, but, if desired, our object may also be accomplished on the tangent screen in the following manner. Have the patient point his finger stiffly beyond him; he will be able by ocular kinesthesia to fixate upon his finger tip, whether he sees it or not;[8] go behind the patient and sight from his eyes along his finger tip, guiding the finger until it points at the marked fixation disc; instruct the patient to maintain this ocular position; move to the screen, and pass a 2 cm. disc outward from the fixation point until he exclaims he can just see it (edge of scotoma); fasten the auxiliary fixation spot to the screen here and instruct the patient to keep it in sight (anchored) without shifting his eyes; then plot the fields as if the central fixation point were actually being used. Other methods may also be utilized, but this is probably the simplest.

INTERPRETATION

The principal clinical necessity for perimetric exploration lies, of course, in the differentiation of conditions amenable to surgical treatment from those whose regimen is wholly medicinal or supportive. The usefulness of perimetry in diagnosis is far more than this, but it is as a means of early detection of surgical conditions that it becomes tragically imperative. Any degree of primary optic atrophy, choked disc, or progressive failure of clarity and acuity despite refractive correction, deserves perimetric exploration. If such a rule were sedulously observed, regardless of full appointment schedules, less hedging concerning belated neurosurgical intervention would be necessary.

Many noxæ may produce optic neuritis, retrobulbar neuritis, or primary optic atrophy, with altered acuity and fields; insidious poisonings with lead, thallium, industrial solvents, aromatics ("ginger"), alcohols (methyl or ethyl), or tobacco, may produce amblyopia with or without neuritis[6] and subsequent atrophic pallor; systemic diseases, such as lues or multiple sclerosis, may similarly affect visual paths. A healed mild secondary optic atrophy (subsided intracranial tension, from any cause, cerebral trauma, compensated hydrocephalus, diabetes, nephritis, polycythæmia, encephalitis, etc.) with amblyopia, may be mistaken by the unwary for a primary optic atrophy unless recognized criteria for ophthalmoscopic differentiation of the two are carried in mind in detail. In general, the perimetric defects produced by the first-mentioned group are vague irregular scotomata, often central or cæco-central and therefore considerably impairing acuity. The second group usually shows concentric constriction of fields, and acuity often suffers little until the final stages. A third group, the defects due to mechanical pressure (or destructive invasion), usually produces some type of true hemianopic defects—bitemporal or homonymous, complete or sector, relative or sharp. It is the detection of the members of this third group which prompts this paper.

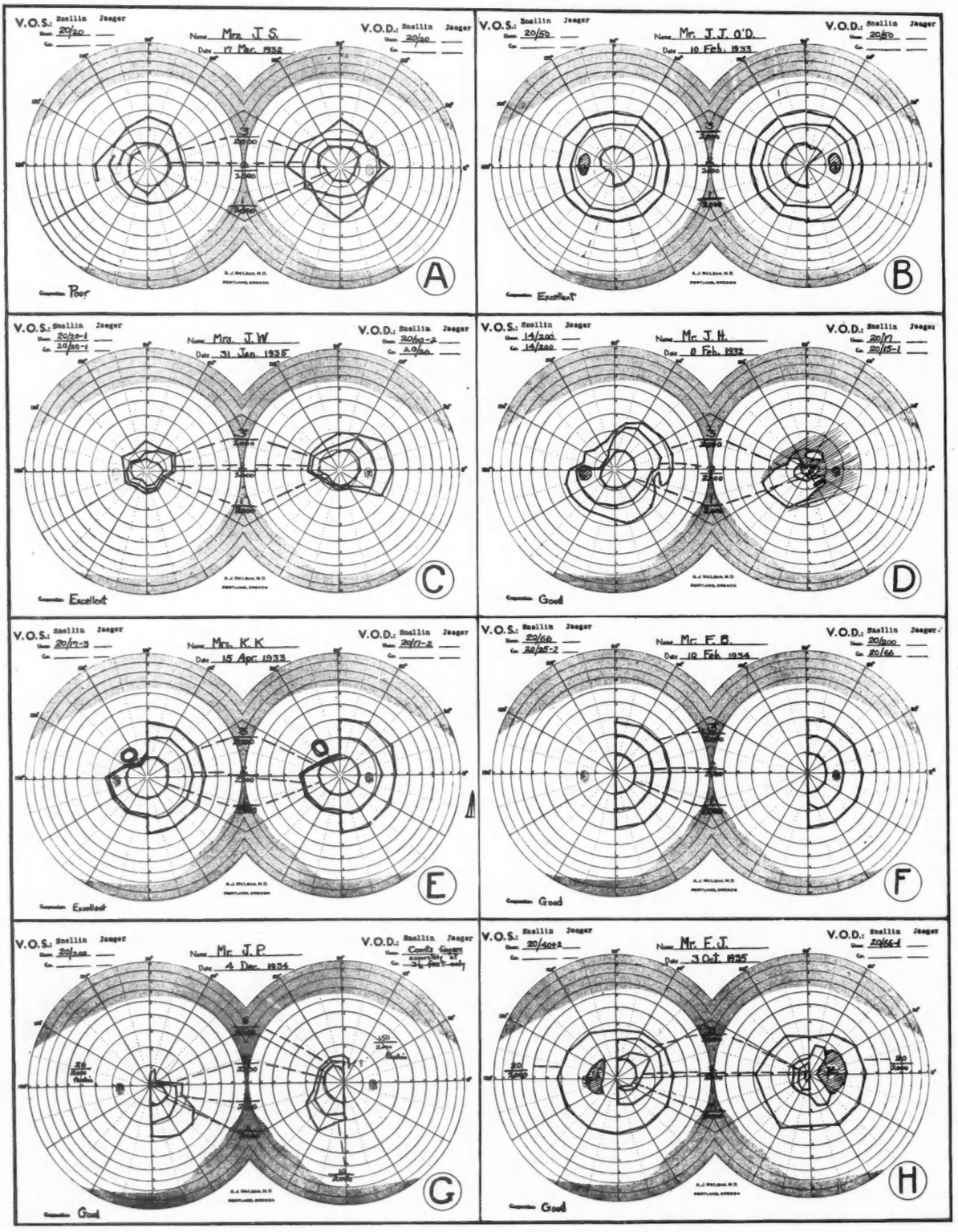
Mechanical pressure upon optic pathways produces first stimulation,* second, physiological interruption, and finally morphological degeneration; the sequence is fortunately considerably separated in time-dimension in cases of localized neoplastic pressure. Since the optic nerve is really a tract of the brain and possesses no neurilemma it is incapable of regeneration, and hence the actual morphological destruction of any of its fibres is irretrievable.

One can obtain some inkling of the degree and type of damage not only by the form of field†

* A word concerning visual hallucinations may not be amiss. Pathological stimulation of the retina or of pregeniculate pathways produces darting scintillæ, infrequent jagged bursts and flashes, or drifting luminous phosphenes. Stimulation of the occipital pole of the calcarine cortex (macular representation) causes hallucination of a ball of light straight in front and stationary; stimulation of the calcarine cortex anteriorly gives rise to moving balls or flashes of light in the periphery, approaching centrally. Stimulation of the visual association cortex (Area 19 of Brodman) gives rise to relatively prolonged hallucinations of flames or of objects (flowers, people, tables, scenes), usually approaching, and with a small amount of (mannikin-like simplicity) motion. It is doubtful whether the Liliputian hallucinations[3] sometimes seen in the hemianopic fields of temporal or parietal lobe tumours actually arise from excitation of the optic radiation (as supposed), or from the adjacent visual association cortex. Complicated organized visual hallucinations, envisaging purposeful action, stem from the frontal lobes. I recall a case of frontal tumour in which the woman thought she saw an ape in a woman's room across the street, and that the ape pulled down the window shade before raping the woman; this had been erroneously interpreted as an occipital lobe symptom.

† As the result of comparatively recent investigations a quite convenient anatomical generalization has become evident. The inferior half of the retina remains representatively inferior in the optic nerve, chiasm, optic tracts, optic radiation, and calcarine cortex; the superior half of the retina remains likewise anatomically superior in representation throughout. This is often of considerable help in localizing lesions at the operating table.

but also by the degree in which successive isopter lines are separated. In general, early physiological interruption is evident by the fact that larger stimuli are capable of penetrating past the barrier, and consequently isopter lines show considerable separation. But when fibres have undergone morphological destruction, no stimulus whatever can pass, and hence isopter lines at the border of such defects are practically coincident (Charts C and E). While this is generally true, it alone is not infallible, for there is not infrequently a complete restora-



Charts A to H.—Visual fields in various pathological conditions; elucidation is given in small print in the text.

tion of vision following the removal of a pituitary adenoma, even in cases which show sharp-line complete hemianopsias; the same also obtains in some temporal lobe meningiomas. Thus, a lag, capable of recovery, still exists for a period of time even after apparent complete coincidence of isopter lines.

Some illustrative fields are shown in the Charts.

A. *Hysteria.*—Here, due to lack of attention, the various isopter lines interweave; this "lacing" is no more characteristic of hysteria than is the "spiraling" shown in the left eye fields, and indicates only rapid fatigue of attention or interest. The findings are significant, but must be evaluated with the case history.

B. *Tobacco-alcohol amblyopia.*—Accompanied by a "wet" fundus ophthalmoscopically, without evident tinctorial alteration, this chart shows the considerable diminution of acuity (despite minor impairment of fields) rather characteristic of neuritides. Exploration with a 0.5 mm. disc might have shown a cæco-central scotoma extending inferiorly from the blind spot to the macula, as the plotted fields suggest. The fields alone, however, were sufficient to rule out tumour. Five weeks of abstinence, with a high vitamin diet and balanced full menu, sufficed to return vision to normal.

C. *Tubular vision* is here accompanied by marked secondary atrophy of optic discs. Medical students are prone to forget that concentric constriction of fields occurs in such conditions; their unfailing answer to a question concerning the significance of concentric contraction of fields is "hysteria", as abetted by many neurological texts.[2] Note the excellent acuity, despite the small fields.

D. *Glioma of the chiasm* is characterized by a rapid asymmetric irregular loss of field, having not a vestige of hemianopic relationship; a relatively firm-founded diagnosis of this condition is warranted by several fields plotted at monthly intervals, showing progressive asymmetric loss. The surgeon is glad of the forewarning, for consultation with parents concerning warrantable procedure.

E. *Cerebral thrombosis.*—Note the perfect homonymous picture, the sharp coincidence of the isopter lines indicating the morphological destruction occurring in the optic radiation, following thrombosis of one of the main posterior branches of the right middle cerebral artery; the lesion and fields (including the retained "island" of vision) have been stationary for years. A similar homonymous quadrantic defect, with spread isopter lines, is often found in temporal or parietal lobe tumours.

F. *Birth injury,* which destroyed completely the right occipital lobe and calcarine cortex; the lesion was demonstrated by encephalography in adolescence; it extended 6 to 7 cm. anteriorly to the occipital pole, and thus accounts for the lack of "sparing of the macula".[1]

G. *Pituitary adenoma.*—The advanced bitemporal hemianopsia was in this instance belied by the minor degree of primary optic atrophy. Consequently, a good prognosis was given, and, post-operatively, the fields and acuity returned completely to normal, and have remained so for two and one-half years.

H. *Cerebellar tumour,* showing anomalous bitemporal hemianopsia due to downward pressure of the floor of the third ventricle upon the optic chiasm from internal hydrocephalus caused by a cerebellar medulloblastoma. Note the spread of the isopter lines, demonstrating that the pressure defect was recent and lightly applied; and the fact that upon using adequately large stimuli (a 20/2,000 disc) the fields were normally rounded. This latter finding allowed confident exploration of the posterior fossa (and removal of the tumour) despite the apparent conflicting bitemporal hemianopsia. The enormous blind-spots bespeak merely the protracted acute papillœdema.

SUMMARY

1. Perimetry is not an esoteric art, but a necessary method of diagnostic exploration of ophthalmological and neurological troubles.

2. The Bjerrum tangent screen, with quantitative perimetry, is an inexpensive, accurate, comfortable method of routine examination.

3. Non-technical details for the construction of a satisfactory screen and for its operation are given; a bibliography, for interpretation, is appended.

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VITAMIN C AND CAPILLARY FRAGILITY.—L. Cotti states that a deficiency of vitamin C causes a capillary fragility which is manifested not only in well-marked scurvy but also in the so-called prescorbutic state or latent scurvy which is revealed by the ordinary tests for capillary resistance, such as the lace test and Hecht's cupping-glass test. Dalldorf and Russell have recently found that patients with well-marked capillary fragility show a rapid increase of capillary resistance as estimated by Hecht's test one to two hours after an intravenous injection of 50 to 100 mg. of pure vitamin C. Similar results were obtained by Cotti both with Hecht's test and the lace test. As entirely identical results were obtained both in normal conditions and in the hæmorrhagic diathesis, he concludes that vitamin C produces an increase of capillary resistance.—*Policlinico,* Sez. Med., March 1, 1937, p. 134. Abs. in *Brit. M. J.*

EXPERIMENTAL GAS EMBOLISM: I. INTRAVENOUS AIR EMBOLISM

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Introduction

THE general problem of embolism has been under study by the Department of Medical Research. Experimental gas embolism, one of the aspects of this problem, has been investigated by the authors, and will be presented in a series of papers. The purpose of this first paper is to consider the relationship between the rate of injection of air and the time necessary to produce death when air is administered intravenously.

Gas embolism has become an increasingly prominent subject due to the larger number of cases being diagnosed following criminal abortion. However, many other procedures may precede this condition. Intravenous gas embolism has been reported following almost every type of surgical operation, particularly thyroidectomy and other operations on the neck. The condition also occurs on the arterial side of the cardiovascular system, usually following therapeutic pneumothorax in tuberculous patients.

In 1934 Harkins and Harmon[1] determined the minimum lethal dose of intravenous air in the anæsthetized dog. This was found to be approximately 8 c.c. per kilogram of body weight when the total amount was injected within 20 to 30 seconds. If this figure were applicable to human cases it would require 480 c.c. of air entering the venous system within 20 to 30 seconds to cause death in a person weighing 60 kgm. It is extremely difficult to conceive of this large amount of air gaining entrance to the venous circulation in such a short period of time. For this reason the effect of varying rates of injection and the relation-

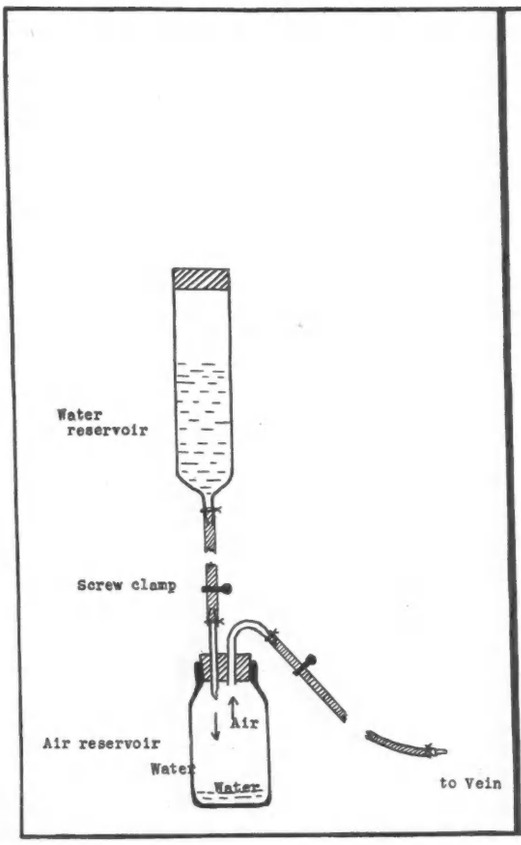


Fig. 1.—Air injected by water displacement.

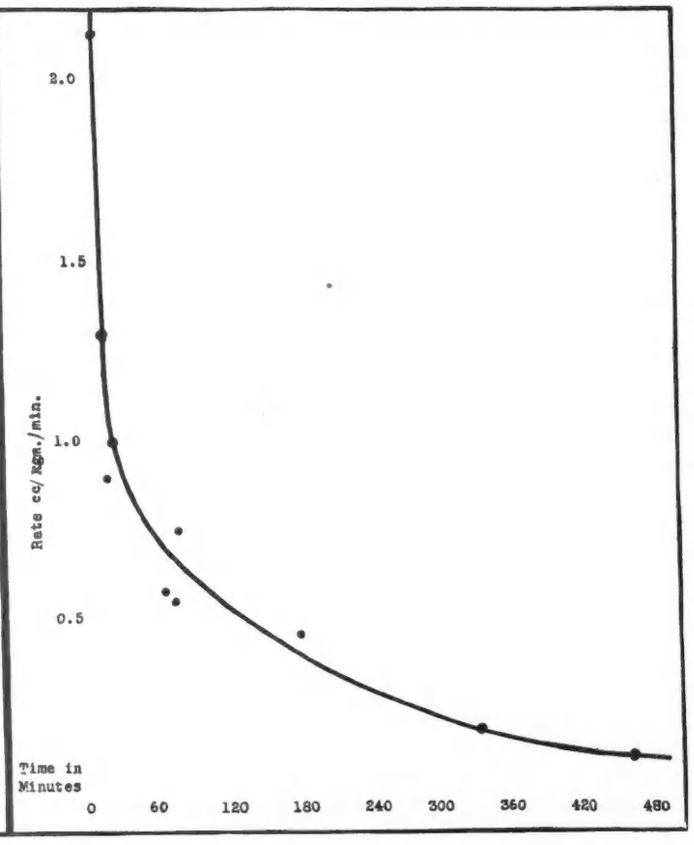


Fig. 2.—Rate of injection (c.c./kgm./min.) plotted against time (min.) during which air was injected.

ship of these rates to the time necessary to cause death were studied.

EXPERIMENTAL

Anæsthetized animals.—Animals were anæsthetized with nembutal (30 mgm./kgm.); kymographic records were taken of blood pressure (recorded from carotid artery), respirations (tracheal cannula), and heart-rate (tonometer connected to carotid cannula). Air was injected through a trocar needle which was tied into the femoral vein. The air was discharged into the vein from a graduated, air-filled reservoir by water displacement. The rate was readily adjusted by a screw clamp controlling water flow into the reservoir (see Fig. 1).

Ten dogs were used in this experimental series. The rate of injection of air was kept constant throughout each individual experiment, and in the series the rate was varied from 0.12 to 2.2 c.c. per kgm. per minute. The results of this series of experiments are shown in Table I.

TABLE I.

Dog No.	*Wt./kgm.*	*Total air*	*Total time*	*Rate (c.c./kgm./min.)*
		c.c.		
7♀*	23.5	3910	87 hrs.	0.033
20	23.0	1377	460 min.	0.12
40	16.0	1056	330 "	0.20
45	8.0	990	180 "	0.47
35	17.0	650	70 "	0.55
60	12.0	455	65 "	0.58
63	13.0	778	79 "	0.76
82	9.0	114	15 "	0.90
47	7.0	140	20 "	1.00
65	5.4	98	14 "	1.30
26	10.0	150	7 "	2.14

*D 79—Unanæsthetized. Time interval beyond limits of graph.

Varying rates of injection in anæsthetized dogs.

It will be seen that there is a definite relationship between the rate of injection and the time necessary to cause death. This is more easily visualized when the rate of injection is plotted against time (see Fig. 2). By increasing the rate of injection from 0.3 to 0.9 c.c. per kgm. per minute an inverse relationship is found between the rate of injection and the time necessary to produce death. When the rate of injection is increased from 1 to 2 c.c. per kgm. per minute the total injection time is almost unchanged. Extremely large volumes of air can be injected at very slow rates. Injecting air at the rate of 0.12 c.c. per kgm. per minute, one dog, weighing 23 kgm., received 1,377 c.c. of air in 460 minutes before death occurred.

From this series of experiments it was concluded that when air was injected at a rate less than 1 c.c. per kgm. per minute fairly large quantities could be tolerated. When the rate was greater than 1 c.c. per kgm. per minute the tolerance was markedly decreased.

The results of sudden injections of air (where a volume of air was injected within 20 to 30 seconds) were then studied. An example is given.

D 11/36 (12 kgm., nembutal 30 mgm./kgm.) was prepared in the usual manner for recording respirations, heart-rate and blood pressure. Air was injected by syringe into a cannulated femoral vein; the injections were given as rapidly as possible. At intervals of 15 to 30 minutes sudden injections of 2.5, 5, 10, 20, 30, 50, 60 and 100 c.c. of air were given. With injections up to 30 c.c. the respirations increased in rate and amplitude, preceded by a transient fall in blood pressure (see Fig. 3). The hyperventilation continued long after the blood pressure had returned to normal. With the fatal injection (100 c.c.) the blood pressure fell promptly to 25 mm. Hg., then tapered off to 0. At the same time breathing became dyspnœic, later apnœic in type, followed by several asphyxial gasps (see Fig. 4). Other experiments confirmed these findings.

In another group of experiments the right ventricular blood pressure was recorded. These experiments were carried out with open-chest technique. It was found that even when death was delayed some hours, although the systemic blood pressure decreased markedly, the pulmonary blood pressure increased from about 10 to as high as 50 cm. of water. It was observed that at death the right ventricle was greatly dilated and the left ventricle was contracted in systole.

Unanæsthetized animals.—In another series of experiments air was injected intravenously into unanæsthetized dogs. Under a local anæsthetic (novocaine) a soft rubber catheter was inserted into the external jugular vein, the tip being kept above the junction of this vein and the subclavian vein. The free end of the catheter was connected to the air reservoir. By means of the swivel unit (Manning, Hall

and Banting[2]), the animal was allowed complete freedom in its cage while receiving the intravenous injection.

Dog D 79 (23.5 kgm.) received 3,910 c.c. of air over a period of 87 hours before death occurred. The injection rate was kept constant at 0.033 c.c. per kgm. per minute.

Dog D 28 (8 kgm.) received 100 c.c. air in 6 minutes (2 c.c./kgm./min.). The animal salivated, coughed, vomited and collapsed; dyspnœa was very pronounced. Even without a stethoscope splashing sounds could be heard as the air and blood were churned up in the right heart. Recovery occurred within 3 to 5 minutes. The following day 300 c.c. of air were injected in 60 minutes (0.63 c.c./kgm./min.). A similar but much less severe clinical picture was noted with this lower rate. Recovery occurred within 5 minutes and the animal remained in good condition. Twelve weeks later it was used as a normal dog in another (unrelated) experiment.

Other dogs received injections of air at rates comparable to those used in the anæsthetized series. It was found that slightly less air could be tolerated by unanæsthetized animals.

The results of sudden injections of air in unanæsthetized animals are shown in Table II. In all cases varying degrees of collapse and dyspnœa occurred. Pronounced bradycardia, cardiac irregularity and water-wheel murmur were constant findings. This clinical condition persisted for four or five minutes in the four animals which recovered. In D 11/37 an asphyxial type of death[1] occurred five minutes after the injection of air. D 7/37, on the other hand, in spite of recovery from the acute condition, still showed signs of pulmonary œdema, which five days later terminated in death from bronchopneumonia. D 8/37 and D 10/37, animals which eventually recovered, showed signs of pulmonary œdema for twenty-four hours.

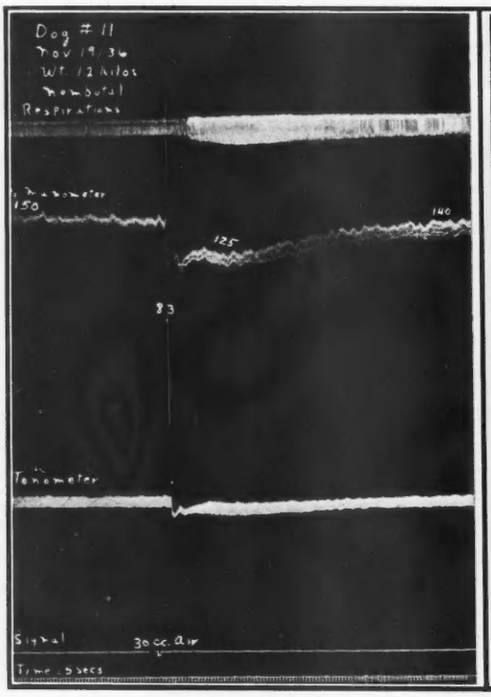


Fig. 3

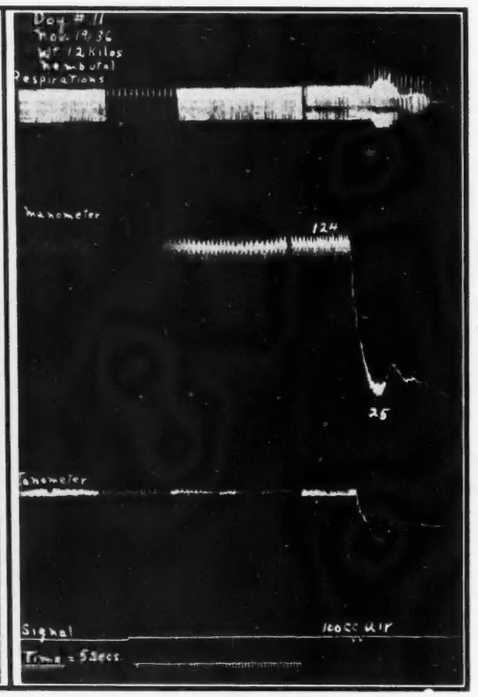


Fig. 4

Fig. 3.—D 11. Nov. 19/36. Wt. 12 kgm. Nembutal anæsthetic. Effect of injection of a small amount of air intravenously (2.5 c.c./kgm). Note transitory fall in blood pressure followed by increase in rate and amplitude of respiration. **Fig. 4.**—D 11. Nov. 19/36. Wt. 12 kgm. Nembutal anæsthetic. Effect of injection of a large amount of air intravenously. Same animal as Fig. 3. Note asphyxial type of death.

TABLE II.

Dog No.	*Wt./kgm*	*c.c. air/kgm.*	*Time in secs.*	*Results*
D 7/37	7.0	5	15	Delayed death
D 8/37	11.3	5	15	Recovery
D 10/37	12.0	6	15	Recovery
D 11/37	11.75	6	25	Immediate death
D 12/37	12.25	6	20	Recovery
D 13/37	12.26	6	25	Recovery

Sudden injection of air in unanæsthetized dogs.

PATHOLOGY

Complete thoracic autopsies were performed. All the vessels going to and coming from the heart were clamped. The trachea was divided and the heart and lungs removed intact. Complete and careful dissection of the heart and lungs was carried out under water.

In all cases air was found in the inferior and superior vena cava, right auricle, right ventricle and the pulmonary arteries. In no case was air demonstrated in the left heart or systemic arteries. In the right heart the air was in a churned-up frothy state, and occasional flecks of fibrinous material were found entangled around the chordæ tendineæ. In three cases a clot of blood and air was found possibly acting as a ball valve against the pulmonary orifice. These clots were about the size and shape of a pecan nut and had the consistency of lung tissue, although more friable.

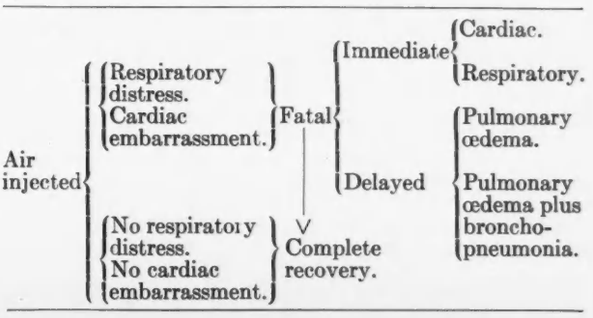
The lungs were heavy and œdematous but still crepitant. The cut surface oozed serous fluid but did not bleed as freely as the normal lung. The bronchioles and bronchi contained much clear frothy fluid.

On section the lung tissue was œdematous. The capillaries were filled with red blood cells, possibly the result of the marked transudation of fluid. In cases in which death occurred more rapidly the œdema was much less marked, although frothy fluid was always found in the bronchi and bronchioles. In these cases too, there was a concentration of red cells in the capillaries and an absence of cells in the veins.

Great care had to be exercised in removing the heart and lungs, otherwise air was readily forced into the coronary veins back through the coronary sinus from the right auricle. Frequently in our experiments air was found in the coronary and internal mammary veins. This has also been reported[3] in coroners' cases. It has been suggested that the air entered these veins after passing through the pulmonary and systemic capillary beds. However, it may be explained as being due to the increased pressure in the right heart forcing air directly back into these vessels. Experimentally, when the right ventricular pressure rises as a result of the obstruction to pulmonary circulation, air is forced back into the coronary sinus and venæ cavæ; from these locations, peripherally into the coronary, internal mammary and even the superficial veins of the chest.

DISCUSSION

From these experiments we suggest the following classification of the effects of experimentally-produced intravenous air embolism:



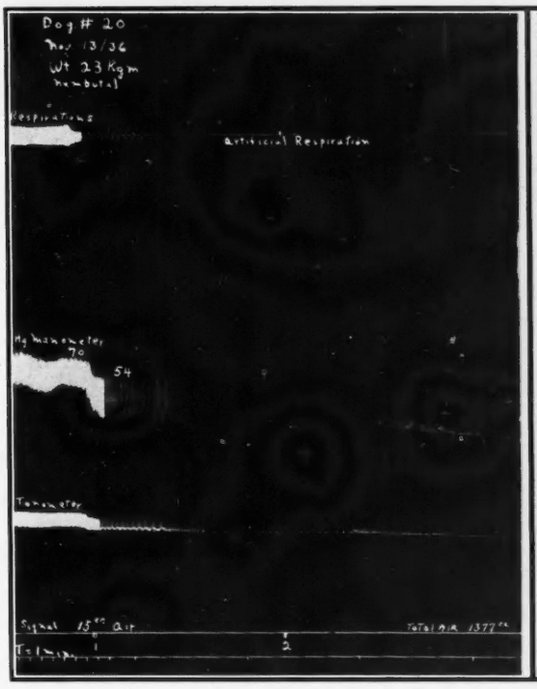


Fig. 5

Fig. 6

Fig. 5.—D 20. Nov. 13/36. Received air at the rate of 0.12 c.c./kgm./min. Total air injected was 1,377 c.c. At signal No. 1, the animal being in very poor condition, 15 c.c. of air was injected. Respirations ceased in 2 minutes. Heart continued to beat for 5 minutes. In spite of artificial respiration (signal 2) the animal died. **Fig. 6.**—D 28. Feb. 25/37. Effect of sudden injection of 64 c.c. of air (8 c.c./kgm.) of a dog with low blood pressure from previous operative procedure. Note the sudden fall in blood-pressure with respiration continuing, as compared with definitely respiratory type of death in D 20.

Obviously, following the intravenous administration of air, the animal either dies or recovers. When the amount of air injected is small the animal usually shows no signs of respiratory or cardiac distress. When larger amounts are injected, either slowly or rapidly, respiratory distress and cardiac embarrassment are evident, but the animal may survive and recovery be complete. On the other hand, the dyspnœa and cardiac embarrassment may be of such magnitude that the injection proves fatal. This fatal issue may be either immediate or delayed. When the injection of air is very rapid (Fig. 5) respiratory failure predominates and death occurs almost immediately; if sufficiently large amounts are given instantaneously acute cardiac dilatation occurs and death is extremely rapid (Fig. 6). In other cases when the air is injected at an intermediate rate, or when a moderate amount of air is injected suddenly, respiratory and cardiac symptoms are evident. Although partial recovery takes place, the animal may develop acute pulmonary œdema and die within a few hours. A bronchopneumonia may become superimposed upon an otherwise non-fatal pulmonary œdema, resulting in death after several days.

Some anæsthetized animals, following the injection of air, tend to hyperventilate themselves very markedly. These animals can tolerate larger quantities of air than other anæsthetized animals whose respirations are less markedly affected.

We feel, too, that the general condition of the animal, as evidenced by its systemic blood pressure, plays a great part in its tolerance to intravenously injected air; much less air is fatal to an animal with a low blood pressure than if the blood pressure is normal. This, we think, is of paramount importance in considering the amounts of air which apparently prove fatal in cases of criminal abortion with which there is associated trauma, pain, apprehension and loss of blood.

SUMMARY

1. The lethal intravenous dose of air decreases as the rate of injection increases.

2. Surprisingly large volumes of air can be injected intravenously without proving fatal.

3. Experiments indicate that anæsthesia increases the dog's tolerance to intravenous air.

4. The pulmonary capillary bed acts as an effective barrier to intravenous air.

5. Death occurs from failure of the pulmonary circulation.

6. The general condition of the animal, as indicated by blood pressure, appears to have a marked influence on the animal's tolerance to intravenous air.

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A CASE OF CONGENITAL MALFORMATIONS OF VESSELS OF THE BRAIN AND SPINAL CORD*

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CUSHING and Bailey[1] and Dandy[2] have pointed out the importance of anomalies of cerebral vessels from the neurological surgeon's point of view. The usual clinical manifestations of congenital dilatation, if and when they become apparent, are such as to lead usually to a diagnosis of cerebral tumour. In many cases, however, the location of these distended vessels is such that no neurological manifestations are noted unless rupture occurs. This is particularly true of those anomalies which involve only the veins. The case which is to be reported gave rise to clinical findings simulating multiple sclerosis.

Present illness.—On July 22, 1935, J.C., a mechanic, aged 36, went to work at 5 p.m. It was a very hot day and he had to work on a hot engine. At 6.50 p.m. he developed a severe occipital headache, became dizzy, and vomited. He was seen by a physician forty minutes later, when he was pale, with a temperature of 100.4° F., pulse, 90, no Kernig or other signs. He was still conscious and vomiting. He became unconscious about

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8 p.m. A second examination at 10 p.m. found him unconscious, with a pulse of 100.

On the morning of July 23rd he was admitted to hospital unconscious. His temperature was still 100.4° F., pulse, 100, respirations, 30. The left pupil was larger than the right, but both reacted to light and accommodation. Rigidity of the neck was present at this time, and movement of the head obviously caused pain. Kernig's sign was present and all reflexes were increased. The discs were pinker than normal. Other organs and systems were normal. Lumbar puncture showed 1,080,000 red cells per c.mm. in the cerebrospinal fluid. Lumbar puncture lessened the depth of the patient's coma.

On July 25th his systolic blood pressure was 126. On July 26th lumbar puncture withdrew fluid under increased pressure. From the evening of July 24th until his death at 8 p.m. on the 26th, his temperature, pulse, and respirations gradually rose to temperature, 106.3° F., pulse, over 150; and respirations, 64. During his stay in hospital he perspired freely and was cyanosed.

A urine examination done on July 23rd was normal. A report on the spinal fluid removed on the day of his death showed it to contain fresh blood. It was sterile on culture and the Wassermann test was negative.

Past history.—The patient served overseas, and received a slight facial wound, to which he attributed many of his later difficulties. Following the armistice he was discharged as fit. He made application to the Re-establishment Board for a business course, which was granted. His work at the College was satisfactory, but suddenly, without cause or warning, he ceased to attend the course, although it meant the loss of his pay and allowances. There is, however, a satisfactory work record between 1920 and 1935, despite certain physical handicaps.

A report of a pension board examination on November 14, 1934, is quoted herewith. "The marginally named man gives a very indefinite history of his present disability. He received two small and obviously superficial gunshot wounds to the right face which he feels have something to do with his present condition, inasmuch as that in 1920 he first noticed some weakness and stiffness in the right leg which at the time he attributed to a callosity of the foot. About 2½ to 3 years ago, the right arm and leg commenced to give him trouble by reason of weakness and stiffness and vague aches and pains. This condition, he states, gradually became worse and more disabling to him. No history is obtainable that would suggest the onset following any trauma, toxic, infectious, or vascular degenerative state. No history of headache, diplopia, bladder or rectal disturbance or alteration in sleep rhythm.

"General appearance is that of a fairly well developed and nourished man who appears at least 10 years older than stated age. Manner is quiet, courteous and cooperative, emotionally a little unstable and reactions at times of the childish type. He presents a slight spastic weakness in the right lower face, arm and leg, with typical increased tendon reflexes, absence of epigastric abdominal and cremasteric reflexes on the right side, and a definite extensor plantar response. The whole body shows a vasomotor instability. Face greasy type, marked dermographia, etc. Musculature is fairly well developed with a definite spastic weakness in right arm and leg. Co-ordination tests performed fairly quickly and accurately. No Rhombergism. On standing, there is a noticeable droop in the right shoulder and a moderate scoliosis in spine, of a compensatory type. No abnormal movements noted. Coarse tremor present in extended hands. Movements of right hand show at times a tendency to the intention type of tremor.

"Both pupils are dilated, the left more so, but both react well to light, although rather sluggish to accommodation. Visual fields appear full. Discs negative except for a suggestion of paleness in both upper temporal fields. There is a poorly sustained coarse lateral nystagmoid movement of the eyes. Mastication, swallowing, speech, etc., normal. Tongue protruded in midline. Some deafness but no tinnitus or vertigo. Sensation. Patient exhibits a definite hypalgesia over the dorsal roots 4 to 6 and lumbar roots 1 to 5 and the lower neck on right side."

The history and physical findings are those of atypical disseminated sclerosis. The clinical findings do not suggest lues. On July 29, 1933, the spinal fluid Wassermann was negative. Measurement of the limbs, done on March 23, 1934, showed three-quarters of an inch of wasting of the right upper arm and slight wasting of the right lower extremity. The right radial pulse was noted to become gradually weaker until it disappeared as the limb was elevated.

Summary of the clinical findings.—The clinical findings show that as early as 1920 there was some weakness of the right arm, and later of the right leg. Latterly, coarse tremors developed in the right hand and right leg, with definite weakness and a patchy sensory disturbance. The paresis was somewhat spastic in type, and there was some wasting. Mentally, childishness and emotional instability were noted. The terminal illness was typical of intracranial hæmorrhage. Cushing and Bailey cite a case of cerebral venous angioma reported by Worster-Drought and Ballance[3] in which the clinical findings led to a diagnosis of multiple sclerosis.

AUTOPSY.—*Gross examination.*—There were no significant findings in any of the organs other than the brain and spinal cord.

Brain.—The brain appeared large. The subarachnoid space showed diffuse hæmorrhage throughout. The gyri were tense and swollen, but there was no evidence of scars or atrophy. The base showed no evidence of gross abnormality. The vessels were not sclerotic. On dissection of the basal vessels, a number of large aneurysmal veins were found, as well as anomalies in the distribution of the basilar artery (Fig. 1). The basilar artery, after giving off the anterior inferior cerebellar branches, broke up into a number of terminal branches which supplied the brain stem. The enlarged posterior communicating branch of the internal carotid arteries was prolonged backward to form the posterior cerebral arteries. Lying beneath the arteries there was noted a number of large aneurysmal veins which appeared to enter the middle of the left cerebral peduncle, while the distal portion disappeared into the substance of the left cerebral hemisphere.

The upper portion of the cerebral hemispheres was removed, exposing the lateral ventricles, which were found to be filled with blood clot. The left lateral ventricle was considerably larger than the right. Death was due to leakage of blood from these aneurysmal veins into the descending horn of the left lateral ventricle. The tip of the left temporal pole was dissected in order to expose the structures to better advantage (Fig. 1). The brain substance in the region of the insula was blood-stained and softened. The left middle cerebral artery was followed to its terminal branches. The greater part of the insula appeared to have been replaced by a mass of dilated veins, the walls of which were very thin, tearing with very great ease. The surrounding brain tissue was soft and very easily broken, and was blood-stained. The mass of dilated veins was dissected and found to be separate from the arteries. They were deeply embedded at their inner end in the tissue of the left cerebral peduncle. The posterior cerebral artery on both sides, which we noted coming off the internal carotid, had no connection with the basilar artery, and so the Circle of Willis was incomplete. The apparent piercing of the peduncle on the left side proved to be only deep embedding of the aneurysmal structures into the peduncle.

Spinal cord.—The dura was not present on the specimen. A transverse section made at the level of the sixth cervical segment showed an area of discolor-

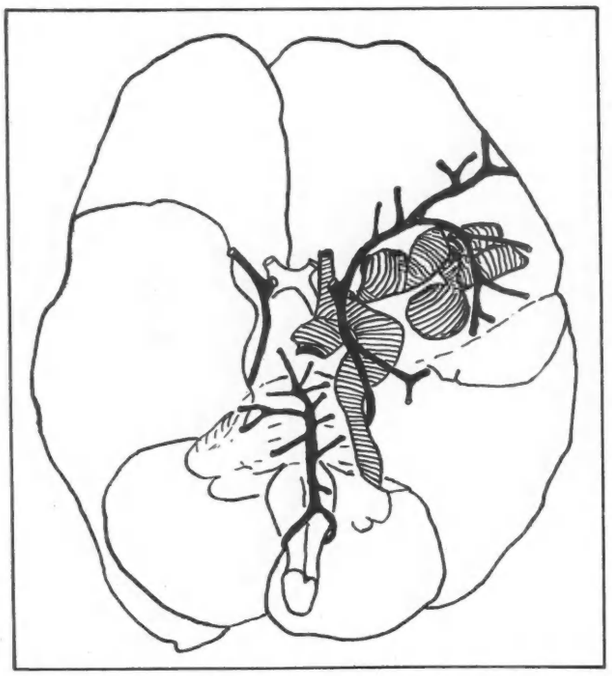

Fig. 1.—The base of the brain after removal of the left temporal pole, dissected to display the aneurysmal distension of the venous channels of the left lateral fissure and brain stem. These veins lay on a deeper plane than the arteries, which were anomalous in their course and distribution.

(This diagram has been prepared as the photographic reproduction of the specimen did not show the details clearly enough). Arteries in solid black; dilated veins shaded.

ation in the anterior horn on the right side. At about the level of the seventh cervical segment on the left side there was another small area of discoloration in the anterior horn. Blocks were taken from these areas.

MICROSCOPICAL FINDINGS.—There was no microscopical evidence of disseminated sclerosis.

Arteries.—Under the microscope the arteries showed thickening of their medial coats, with an occasional area of early hyaline degeneration. The internal elastic lamina was poorly formed and not as sharply differentiated from the media as usual. The arterial coats were poorly differentiated from each other and were showing degenerative changes, suggesting vessels of congenitally poor formation, especially in view of the fact that the patient was a young man.

Veins.—A section taken of a very distended portion of the left middle cerebral vein showed the wall to be very thin in places and thickened in others (Fig. 2). It was composed in the main of fibrous tissue, much of which showed hyaline degeneration. Rarely, a few muscle-fibres were seen. For the most part the intima was composed of a single layer of endothelial cells.

A block taken from another portion of the vein showed quite a different picture. In the main the wall was thin, but in some areas its medial coat was thickened, with invasion of the media by fibrous tissue from the adventitia, much of which showed hyaline degeneration. Occasionally, the intima showed proliferation, with a tendency toward concentric arrangement of the cells. In other portions, the thickening took the form of what Buckley[4] describes as leiomyomatous nodules. Van Giesen's stain failed, however, to demonstrate muscle-fibres. One part of the vessel showed a primitive type of internal elastic lamina in part of its wall, suggesting that it might be an extremely poorly-formed artery, but these vessels had the histological structure of veins.

A block taken from the tissue of the insula surrounding the distended vein showed the type of vessel just described in very large numbers. There was a marked increase in numbers of endothelial cells, suggesting a persistence of the embryonic type of undifferentiated blood-channels, in some of which there was scarcely any lumen (Fig. 3). Many of these channels could be seen to be filled with blood while others contained hyaline thrombi. The tissue between these primitive blood-channels was degenerate and composed mainly of glial tissue, an appearance which is not found in neoplasms of vascular origin. Fresh and older hæmorrhages were found in the tissue.

A block from the cervical region of the spinal cord at the level of the 6th or 7th segment showed, in the gross, what appeared to be hæmorrhage. Under the microscope, no definite hæmorrhage was found in the anterior horn. There were, however, definite areas of hæmorrhage in the white matter (Fig. 4). The capillaries were more numerous than normal, and many of them were of the embryonic type seen in the tissue of the insu'a. Some of these vessels were very markedly dilated and coiled on themselves, a picture in miniature of the veins seen in the brain (Fig. 5). There were a number of petechiæ in the fibres leading to the posterior horns, but hæmorrhage was mainly confined to the anterior and lateral columns of the white matter of the cord. These hæmorrhages were fresh in the sections examined. Weigert preparation of the lower cervical spinal cord showed patchy areas in which the myelin appeared lighter than the surrounding tissue, although no definite beading could be distinguished. A section from the lumbar region of the cord showed numbers of thin-walled blood vessels and similar light areas in the Weigert stain, although no hæmorrhages could be seen. The vessels in the spinal subarachnoid space showed

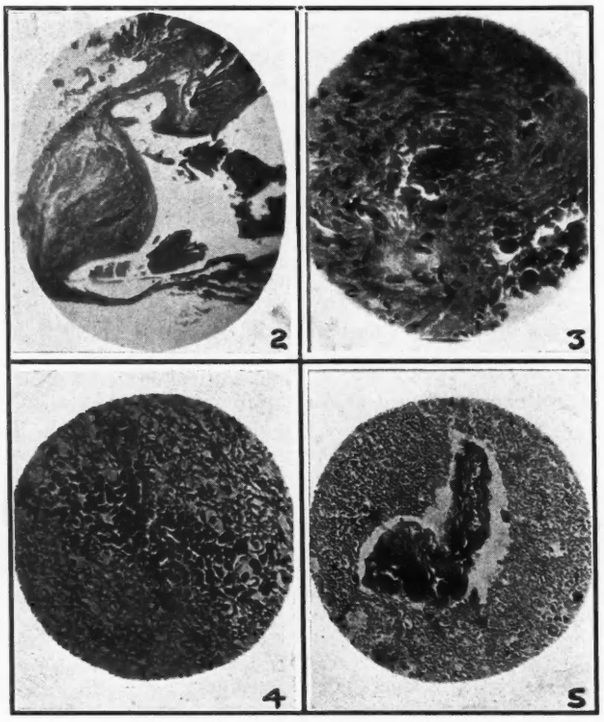

Fig. 2.—A low power view of one of the distended veins. Note irregularity of its wall and probably point of rupture with extravasation of blood into vessel-wall.

Fig. 3.—One of the primitive blood-channels in the insular tissue. Note the large pigment-containing cells indicative of old extravasation of blood.

Fig. 4.—Area of hæmorrhage in the white matter of the cervical spinal cord, at the level of the 6th or 7th segment.

Fig. 5.—Malformed vessel in spinal cord at the level of the 6th or 7th cervical segment.

marked dilatation with increase in their number. The majority of these were thin-walled, but a few showed thickening of a degenerative type.

DISCUSSION

The clinical picture in this case did not fit in with any well recognized nervous disease, and the diagnosis of atypical multiple sclerosis was made. As previously pointed out, there is at least one other case of similar type in the literature in which this diagnosis was made. To assist in an appreciation of the pathological findings in this case, a brief review of the stages of the normal embryological development of the cerebral blood vessels is given, as the lesions found were essentially those of anomalous development of these structures.

G. L. Streeter[5] showed five stages in the development of the blood vessels of the human brain: (1) the establishment of primordial vascular plexus; (2) the primordial plexus slowly resolves itself into arteries, veins, and capillaries; (3) a splitting into a superficial, a dural, and a pial circulation; (4) arrangement into a series of vascular trunks; (5) the development of the adult structures of arteries and veins.

There is little which can occur by way of anomalies in stages one and five, and the anomalies resulting from stage four give rise to gross anomalies of distribution of larger trunks (such as the basilar arteries in this case show). Malformations in the third period could give rise to the peculiar communicating anomalies of the type of telangiectasis, in which certain vascular anomalies of the scalp have been found to be connected with similar structures of the dura and underlying brain. The second period of development, although most frequently being the origin of more malignant types of tumours, may be the origin of benign structures, the differentiation of which into arteries or veins may be difficult, and which may result in gross malformations in advanced life such as has been shown in this case.

As pointed out by Cushing and Bailey, telangiectases are of little importance to the neuro-surgeon, but may bear a definite relationship to mental disturbances. The various types of venous angiomata may give rise to a clinical picture resembling brain tumour, and may therefore be uncovered by the neuro-surgeon. It has been suggested (Cushing and Bailey) that venous angiomata do not become aneurysmal unless they communicate with arteries, but no apparent arterio-venous communication was present in this case, in which distension of the venous walls was very evident. One of the vessels, however, showed an incomplete internal elastic lamina, and appearances suggestive of Buckley's "leiomyomatous nodules" were present in one or two areas, both of which findings suggest tendencies towards an arterial structure. The dilatations of the veins in this case were in such locations in the brain and spinal cord that they produced a picture, through pressure or hæmorrhage, or both, simulating multiple sclerosis.

SUMMARY AND CONCLUSIONS

1. A case of right hemiparesis with patchy areas of anæsthesia has been reported.

2. The cause of these peculiar clinical manifestations can be related to lack of developmental differentiation of the cerebral and spinal veins in the second and fourth stage of fetal development (Streeter).

3. Dilatation of, and multiple hæmorrhages from, these vessels gave rise to a clinical picture simulating multiple sclerosis.

4. The veins mainly affected were those in the left lateral fissure. Rupture of these anomalous, distended vessels, and leakage into the descending horn of the left lateral ventricle caused death in four days.

5. The anomalous distribution of the basal cerebral arteries played no significant rôle in either the clinical findings or death of this patient, but provided further evidence of defective embryological development of the vascular system.

6. This case suggests that venous malformations may become aneurysmal without connection having been established with arteries.

I am indebted to Dr. E. P. Johns, of London, Ont., for the material of this case and to the Department of National Health and Pensions for some of the history. Dr. M. S. Thompson's assistance was most valuable. Prof. E. A. Linell's critical advice was also much appreciated.

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MULTILOCULAR POLYCYSTIC TUMOUR OF THE PANCREAS*

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CYSTS of the pancreas are rather uncommon and are usually difficult to diagnose clinically. In contrast with certain solid tumours of the pancreas, such as adenoma of the islands of Langerhans and carcinoma of the head of the pancreas, cysts of the pancreas seldom disturb the metabolic functions of the pancreas enough to attract clinical attention. They may, however, cause pain and symptoms of pressure on adjacent structures. Most frequently they manifest themselves as an upper abdominal mass of rather limited mobility. In the absence of characteristic symptoms the differential diagnosis may lie between pancreatic cyst, mesenteric cyst, retroperitoneal tumour, occasionally, cyst of the spleen and other uncommon conditions. The gastro-intestinal and urinary tracts may be excluded by roentgenological examination. The exact diagnosis is usually made at the time of exploratory operation, which is generally indicated.

There are five main types of pancreatic cysts: (1) congenital; (2) retention; (3) proliferative; (4) so-called pseudocysts, and (5) hydatid cysts. Probably the three most common types are the retention, proliferative, and pseudocysts. Retention cysts may form when obstruction occurs to some part of the collecting system of the external pancreatic secretions. The main or lesser pancreatic ducts may be involved. Proliferative cysts are neoplastic, and may or may not be highly malignant. Not infrequently they are endotheliomatous in nature, fairly well encapsulated, and can be enucleated without great difficulty. Pseudocysts are not true cysts in the strict sense of the word as they have no definite lining membrane and are usually traumatic or inflammatory in origin. The following report concerns a patient who had a proliferative cyst of the pancreas.

* Submitted for publication, October 19, 1936.

† Now residing in Vancouver, B.C.

CASE REPORT

A woman, 42 years of age, came to the clinic on May 8, 1936, complaining of a "bunch in the stomach" which had been present for four years and which had slowly increased in size. During that period the patient had suffered from attacks of gaseous abdominal distress, accompanied by a burning sensation in the epigastrium. This occurred immediately after meals. Occasionally these attacks were associated with colicky pains in the upper portion of the abdomen which were relieved when the patient lay down. She also had had severe occipital headaches, which were more severe at the menstrual period than at other times, and attacks of dizziness associated with difficulty in walking. A complete neurological examination had been made in 1918 because of these complaints, but negative results had been obtained and the symptoms were thought at that time to be entirely functional in origin.

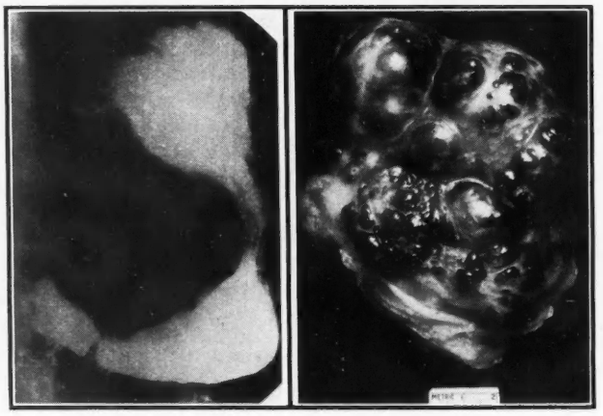

Fig. 1 Fig. 2

Fig. 1.—Roentgenogram showing displacement of stomach to the left. **Fig. 2.**—Multilocular pancreatic cyst following removal.

Physical examination at the clinic revealed a large cystic mass in the left upper quadrant of the abdomen. The mass moved on respiration and did not seem to be firmly fixed. Examination of the eyes and nervous system gave negative results. There was no apparent disturbance in carbohydrate metabolism. Roentgenological studies of the stomach revealed an extrinsic mass which displaced the stomach to the left (Fig. 1).

Abdominal exploration was carried out on May 14th through a left rectus incision. Palpation of the spleen, kidneys and liver did not reveal any abnormality. A multilocular cystic mass, about 10 cm. in diameter, was found presenting above the lesser curvature of the stomach; this mass arose from the tail of the pancreas. It was removed, together with some pancreatic tissue. There were numerous large vessels running into the cyst, and, since it was difficult to ligate these satisfactorily because of their depth in the wound and because of the friability of the tissues, some curved

hæmostats were left in place to control bleeding and were packed off from the rest of the viscera with iodoform gauze. Seventy-two hours later the clamps were loosened and then removed. This was followed by some bleeding, which was controlled by an iodoform pack. Two transfusions of 250 c.c. each of citrated blood were administered on the two succeeding days. The iodoform packs were removed slowly and the wound was closed by secondary suture on June 15th. Her progress from this time on was satisfactory and the patient was dismissed from the clinic in good general condition on July 15th.

The pathologist reported the specimen to be a benign multilocular polycystic tumour of the pancreas, 10 by 9 by 6 cm. in size. It weighed 300 gm. (Fig. 2).

COMMENT

Tumours or cysts of the pancreas may be approached surgically along the lesser curvature of the stomach through the gastrohepatic omentum, or below the greater curvature through the gastrocolic omentum. The latter approach is usually more desirable. It was not utilized in this case, however, because the mass actually was situated beneath the gastrohepatic omentum. In dealing with simple unilocular cysts of the pancreas, if complete enucleation does not seem advisable, drainage or marsupialization is the simplest and safest surgical procedure and one that is usually followed by satisfactory results. Multilocular cysts, however, are likely to be malignant, and, accordingly, complete extirpation is usually desirable.

The pathologist reported that the lesion of the pancreas in this case was a polycystic tumour similar to those found in Lindau's disease. This is one of the less common types of cysts of the pancreas. In Lindau's disease hæmangiomas of the cerebellar region are often associated with hæmangiomas of the retinal vessels and with polycystic disease of the pancreas, liver, kidneys and other organs. In this case some of the symptoms, such as headache, dizziness, and mild ataxia, suggested the presence of a cerebellar lesion. These symptoms, however, had been present for many years and their severity had not increased. Neurological examination did not reveal any localizing signs of a cerebellar lesion and the ocular fundi were normal. The neurological consultants therefore felt that if any hæmangiomas were present in the cerebellar region they were too small at the time to be of any consequence.

THE TREATMENT OF SCOLIOSIS*

BY R. G. HUCKELL, M.D.

Edmonton

IT has been said that lateral curvature of the spine, or scoliosis, is one of the penalties of the upright position. This is not altogether true, but I think the effect of gravity on the curves should be borne in mind. Scoliosis has been classified as to etiology and as to clinical findings. Clinically, we have it divided into two broad classes—the postural or false scoliosis, and the structural or true scoliosis. We might briefly differentiate one from the other.

The postural is one long curve, or a total scoliosis. Ninety per cent of the cases have their convexity to the left, the left shoulder is high, and on forward bending the two scapulæ appear level. The structural, on the other hand, has a compound curve, *i.e.*, there is a curve which is secondary to the primary one. There is definite rotation of the bodies of the vertebræ, and often bony change. On forward bending the scapulæ are of different heights and the ribs on the convexity are prominent as opposed to the postural, where the prominence is on the concave side. The primary curve is always the fixed one and shows rotation.

The late Dr. Lovett, of Boston, made the following statement. "What the prevailing treatment of scoliosis is today cannot be stated in a few sentences. Some men advocate one method, some another. To those of us who believe that forcible correction has accomplished more than any other treatment and will accomplish more in the future the development of the real treatment of scoliosis dates from 1875. To those who believe that the gymnastic treatment of scoliosis is the best and that forcible correction is wrong the progress in the matter

* Read at the Annual Meeting of the Canadian Medical Association, Alberta Division, on September 17, 1936.

of scoliosis starts with Hippocrates, and comes down in a wavering and discouraging line from that date until the present, with a better knowledge of the affection on the whole, with probably some more effective gymnastic technique, but without any very great advance within the last hundred years in the line of efficiency.'' The statement of Dr. Lovett, I think, indicates that there is no one way of treating scoliosis, because it is generally believed now that gymnastics and active exercise are the proper method of treating postural or false scoliosis, and that the best results in structural scoliosis have been obtained by using forcible correction, plus surgery, plus active exercise. When a patient with scoliosis presents herself, and I may add that 75 per cent of scoliosis occurs in girls, the reason for which has not been satisfactorily explained, x-ray plates are taken of the patient standing, and a study is made of these to ascertain the apex of the primary curve. This point is then marked on the skin. Also the angle of deviation is measured. This angle is formed by two lines which are drawn as follows, one from a normal vertebra at the top of the curve joined to the apex, the other, from a normal vertebra at the bottom of the curve to the apex, this line being produced to form what is called the angle of deviation. This is done in order that we may measure accurately the degree of our correction, and not be carried away by an optimistic attitude (Fig. 1).

I propose to outline the method as carried out by Risser and Ferguson, of the New York Orthopædic Hospital, in the treatment of structural scoliosis. This method we have used in some 15 cases since 1931, and have felt that it is a decided improvement on forcible correction alone or gymnastics alone, or on forcible correction plus gymnastics. I may add that the cases which we have handled have been those due to anterior poliomyelitis and those with congenital anomalies, which are said to be the primary cause.

The method consists of the application of a plaster of Paris spica jacket which extends from the occiput to the knee on the convex side and to the trochanter on the concave side. When the jacket is applied no effort is made to make any correction. Hinges are then centred over the apex of the curve, which has been previously noted (Fig. 2). These are incorporated into the plaster jacket both on the anterior and posterior surfaces; then lugs are applied on the concave side with a short turn buckle. After the jacket is hardened, probably the next day,

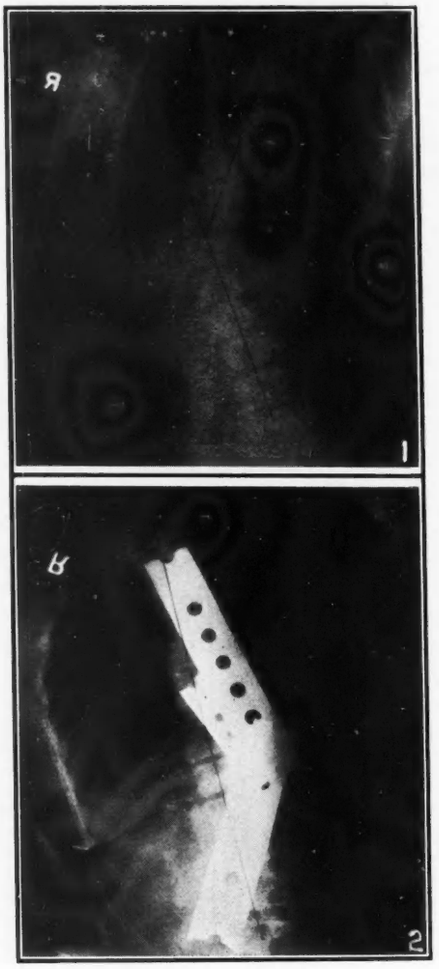

Fig. 1.—X-ray plate of patient standing.
Fig. 2.—Nine days after application of plaster of Paris spica jacket showing curve corrected.

a large ''V'' is cut on the convex side and a straight line cut around the concave side, no attention being paid to the secondary curve. After these areas are cut through, the turn buckle is used to forcibly bend the primary curve over toward its convexity, and longer and longer turn buckles are used until satisfactory correction is obtained, or until correction is as complete as is possible. The jacket is then built up with plaster to strengthen it and hold it in this position. We use splint boards on the anterior surface so that our jackets will not break. A large window is then cut out at the back, to expose the whole area of the primary curve, and a fusion is then

done the full length of the primary curve. On exposing the spine it has been noted that the interarticular facets on the concave side have been spread apart and in our fusion, which is the Forbes' modification of Hibbs', bone chips are forced into this articular facet after the articular cartilage has been removed. It is sometimes necessary in long fusions to do the fusion in two stages, as a fusion of 9 to 10 vertebræ often causes too much shock for the patient to stand at one time. The patient remains in this spica for a period of 3 to 4 months, pain at the site of fusion being considered evidence that bony union is not complete, as x-ray plates often show what appears to be fusion and yet, clinically, fusion is incomplete. After this time the spica is removed and a plaster of Paris jacket applied, extending from the jugular notch to the trochanters. This forcible correction quite often increases the secondary curve, and it is very gratifying after the removal of the spica to see the secondary curve straighten up and note that the back appears straight. This jacket is worn for a period of nine months, during which time the patient is on physical training, developing the back muscles by exercise and doing all the usual gymnasium physical training. The last three months the patient wears a split jacket, getting out of it daily, gradually increasing the time out of the jacket. After this period the jacket is removed.

One might say that this is rather a heroic procedure, but when we consider that structural scoliosis will tend to increase until the person stops growing, and that the patient will be obliged to wear apparatus until that time, I feel that we are quite justified. Condemning a girl of 7 or 8 to a plaster or a steel and leather jacket until she is 17 or 18 years of age, with very little hope of correction and in all probability an increasing curve, I believe is a much more radical procedure than forcible correction, plus surgery, plus gymnastics.

HAY FEVER IN ALBERTA

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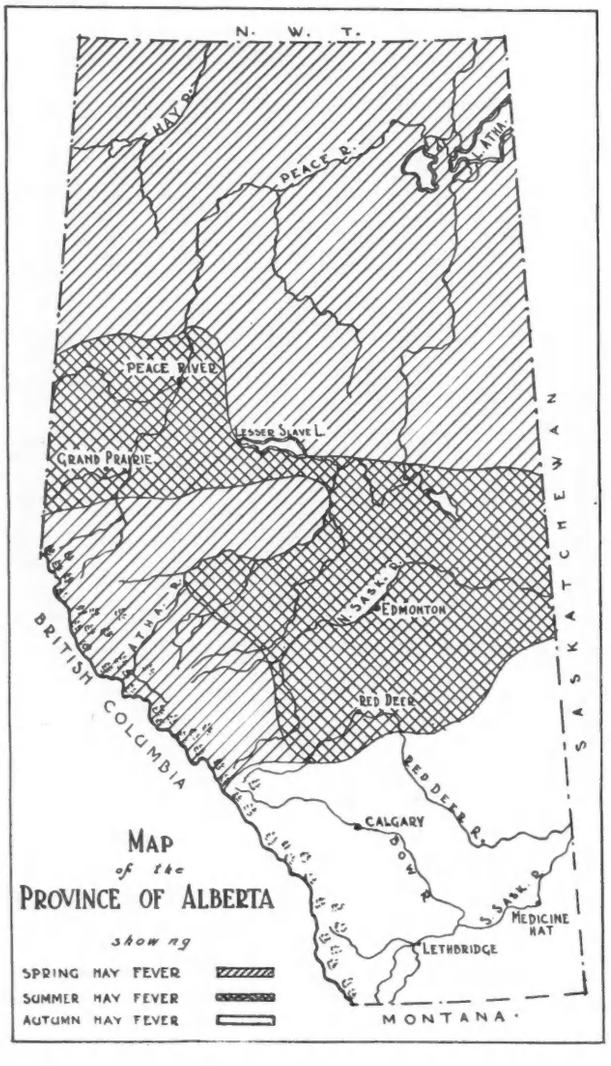
THE diagnosis of seasonal hay fever or pollen allergy is usually made by means of skin tests. These must however be interpreted in the light of botanical studies of the locality concerned. Due consideration must be given to the pollen content of the air as well as to the capacity of such pollen to cause this affection. The pollen allergy problem of Alberta differs somewhat from that of other prairie provinces, and markedly from that of British Columbia and of Montana just below the international line.

The Western Canadian plain from an altitude of from 500 to 1,000 feet in Manitoba rises gradually to an elevation of from 1,000 to 2,000 feet over the greater part of Saskatchewan, to attain a height of from 2,000 to 5,000 feet in southern and central Alberta before losing its identity in the foothills and the Rocky Mountains. The physical character of the province changes from south to north. Between the border and the Red Deer River stretches an undulating prairie. From thence northward to the North Saskatchewan River the land is semi-prairie. Beyond this extends the Northern Forest Belt. In the Peace River district, however, there are large areas of park land. The altitude becomes lower from south to north, dropping to 1,200 feet along the Peace. Extending from 49° N. lat. Alberta stretches to 60° N. lat., a distance of 800 miles, and has an area of 257,000 square miles.

It can be stated broadly that Alberta is composed of three hay-fever zones. One reaches from the Red Deer River to the northern boundary. In this area trees are the chief offenders, and spring is the time of their offensive. A second comprises the southern section of this same region, with its upper limit in the Peace River country and with grasses and summer weeds as well as the spring trees causing trouble. A third, extending from the Red Deer River south to the international boundary, is scourged by the Russian thistle and the sage in the fall. Few trees are to be found on the prairie excepting in the river bottoms, where poplar and willow fringe the streams. On the semi-prairie these two trees

are common. The forests of the north contain poplar, willow birch, spruce and pine. The poplar reaches a higher degree of development in the Peace River country than elsewhere in America.



In order to obtain a clear view of the pollen allergy problem in Alberta certain facts about this subject in general must be presented. Thommen has predicated five postulates in regard to causation.—

1. The pollen must contain an excitant of this condition.
2. The pollen must be anemophilous or wind-borne as regards its mode of pollination.
3. The pollen must be produced in sufficiently large quantities.
4. The pollen must be sufficiently buoyant to be carried considerable distances.
5. The plant producing the pollen must be widely and abundantly distributed.

Three classes of plants must be considered in any analysis of this subject—trees, grasses and weeds. A better picture of the situation can be presented by a brief survey of the common offending plants in North America, and then considering such as are of consequence in Alberta.

A fact of interest and importance gives one a general idea of the hay-fever study of America—the farther north one goes from the tropics, the more the tendency for pollination to be by wind.

Trees.—The tree-groups on the continent which meet the requirement of the five postulates of Thommen are three in number.

1. The catkin-bearing trees: oak, birch, willow, poplar, hickory, walnut, beech, alder and hazel. All of these are wind-pollinated but the willow. In Alberta only the poplar and willow need be noted. The latter is of secondary importance.

2. The trees producing unisexual flowers not in catkins: maple, ash, hackberry, sycamore and mulberry. None of these are indigenous to this province.

3. Trees producing perfect flowers: elm and mesquite. The elm has been introduced as an ornamental tree, but can be disregarded. The mesquite is unknown in the latitude of Alberta.

To sum up: of all these trees the poplar alone is of moment in this study, and then only from the Red Deer River to the northern boundary. This tree and the willow are the first offenders in the spring. The middle of April is the earliest date at which they pollinate. In the foothills and the mountain regions the season may be from ten days to two weeks later than in the eastern parts of the province, so the pollen allergy victim may escape an attack by visiting the Jasper National Park or the Banff National Park. The length of pollination of these trees is often not more than two weeks. A heavy rain just as the pollen is being shed may destroy much of it. A day or two of cloudy weather at the time of pollination often aborts this process or markedly diminishes its amount.

Grasses.—Three grasses are of particular consequence in America: (1) timothy; (2) June grass; (3) Bermuda grass.

Timothy is present in the central area of Alberta and in the Peace River country. June grass is much less in extent. Bermuda grass has never been introduced. Red-top is not native but is found in some districts. June

grass usually pollinates about the middle of June, and timothy during the last week of this month.

Weeds.—The weeds of the continent can be considered in two groups: (1) the ragweed family (Ambrosiaceæ); (2) the thistle family (Compositæ).

Neither the low ragweed (*Ambrosia artemesiafolia*) nor the high ragweed (*Ambrosia trifida*) is found in the province, excepting to a slight extent in the area south of Medicine Hat. In a recent survey of Montana a count of ragweed pollen gave an average of only six grains, which is considered below the number capable of producing symptoms. It is true that the Xanthium branch of the family is represented by the cocklebur in the same area, but this is of minor importance.

The thistle is the largest plant family, but in America only one genus, *viz.*, Artemisia, is of prime interest to the allergist. This is represented in Alberta by the prairie sage and the carpet sage, which are more abundant south of the Red Deer river.

A second group of the family comprises the following: (1) the pigweed family (Amaranthaceæ); (2) the goosefoot family (Chenopodiaceæ); (3) the plantain family (Pantaginaceæ); (4) the buckwheat family (Polygonaceæ); (5) the hemp family (Cannabinaceæ).

The pigweed and the goosefoot need some consideration in Alberta. Red root pigweed (*A. retroflexus*) and the Russian pigweed (*Axyris amarantoides*), first discovered in Manitoba in the eighties, and which has since become widespread in the coarse grain territory of Western Canada, can be found in the central portion of Alberta and in the Peace River district. Two other members of this family, the tumbleweed (*A. græcizaus*) and the careless weed (*A. palmeri*) are of minor importance in the south.

America has several kinds of goosefoot: (1) Chenopodium; (2) Kochia; (3) Atriplex; (4) Salsola. In Alberta the only Chenopodium is the album (Lamb's quarters). This grows most profusely in the central area and the Peace River country. These districts produce the coarse grains and the most profuse weed crops of the province. One other plant in the goosefoot family, the Russian thistle, is a frequent cause of pollen allergy in Alberta. It is confined to the area south of the Red Deer River.

One must always remember that in certain localities plants which are uncommon causes of trouble may be found. For instance in the Edmonton district the willow and dandelion, both insect-pollinated plants, are causes of allergy.

From this study, made after many years of field surveys in all sections of Alberta, from pollen counts in Edmonton and from skin tests, it has been found that the poplar and the willow are common spring excitants of hay fever, particularly north of the Red Deer River. Next in that zone the dandelion is a minor cause. Mid-June brings June grass and late June timothy in the same district. In July and onwards the weeds may be indicted. Russian thistle and sage harass the susceptible in the south until the frost cuts short all plant life.

With the clearing away of the bush-land between the North Saskatchewan and the Athabasca rivers the area of coarse grains is extending northward, and so the culpable grasses and weeds are creeping a hundred miles farther towards the Arctic. Already one can see stands of Artemisia, with specimens over seven feet high, on the banks of the Athabasca.

TRANSITORY HYPERGLYCÆMIA AND GLYCOSURIA IN ACUTE CORONARY OCCLUSION.—K. W. Blake reports his case of transitory hyperglycæmia and glycosuria occurring in a patient with acute coronary occlusion for the purpose of emphasizing one of the frequent clinical features of acute coronary occlusion that is often misinterpreted and mistreated—transitory hyperglycæmia and glycosuria. It is misinterpreted because the condition is apt to be confused with true diabetes mellitus and mistreated because insulin is often employed in such doses that the blood sugar is rapidly lowered, thereby leading to serious faults in conduction, namely, extrasystoles, auricular fibrillation and bundle branch block, to acute congestive failure or frequently to death. That a high level of blood sugar is not deleterious but rather advantageous to the cardiac patient, particularly in the anginal type of cardiac distress, has been the clinical experience of many. As to the cause of the rise in the blood sugar, no one can at this time definitely say, although many theories have been advanced. Whether it is a physiological or a pathological response has not been determined. The analogous occurrence of hyperglycæmia and glycosuria in patients suffering with circulatory shock following cerebral hæmorrhage, embolism and thrombosis may aid in the future in solving this problem. What is known, however, is that in a certain percentage of patients suffering with acute coronary occlusion such a phenomenon does occur and that when it is seen one must remember that it does not necessarily indicate any true diabetic state, that it will usually subside of its own accord, and that the employment of insulin is usually not necessary.—*J. Am. M. Ass.*, 1937, **106**.

COINCIDENTAL DIABETES MELLITUS AND RENAL GLYCOSURIA*

BY E. LOZINSKI, M.Sc., M.D. AND L. I. FROHLICH, B.Sc., M.D., C.M.

Montreal

THE establishment of renal glycosuria as a disease entity, its etiology and diagnosis have formed the subject of many reports in recent years. One of the most comprehensive of these is that by Falta.[1] The condition is relatively uncommon. Thus Marble[2] found among 9,000 cases with glycosuria 17 typical and 7 atypical which conformed with the presently accepted standards for a diagnosis of renal glycosuria. The figures published by other observers show an approximately similar incidence.

On general principles the association of renal glycosuria with diabetes mellitus may be expected. However, the rarity of such an occurrence and the importance of its recognition from the standpoint of treatment occasion this report.

The first reference to a combination of renal glycosuria and diabetes mellitus was made by Vogelenzang[3] in 1925, who reported 2 cases. He stated that up to the time of his article he knew of no analogous cases in the literature. Curran and Mills[4] and Voigt[5] each reported a single similar instance in 1927. V. Holst,[6] in 1932, reported a case he diagnosed in 1930. Winkler[7] reported a case in 1934. He states that Umber and Rosenberg had seen only 37 such cases in fourteen years, and that Falta described 12 mixed cases.

CASE REPORT

Mrs. M. (No. 6499), aged 58, a Jewess, housewife, first came under our observation in July, 1934. Complaints—diabetes.

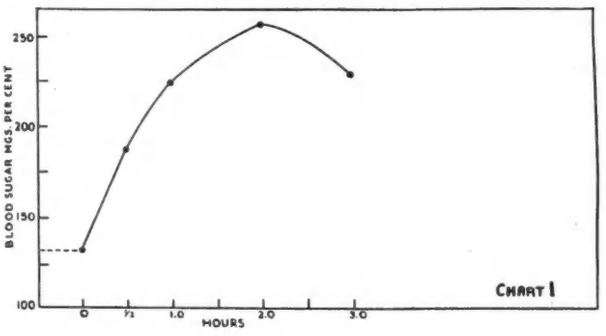
The diagnosis had been made in January, 1933, when she was living in western United States. At that time her complaints were thirst, frequency, generalized pruritus, and loss of weight. She was given a diet of about 1,300 calories, with insulin 25-0-15. Her weight prior to the onset of her illness was 123 lbs. She had no other complaints. The past history was uneventful, except that in 1931 she had a severe emotional episode when one of her sons was drowned. She had never had any serious illness and always enjoyed good health. She married young and gave birth to nine full-term children. There were no miscarriages. Menopause occurred at 49. There was no family history of diabetes.

She consulted us for the purpose of checking her diet and insulin dosage. She was completely symptom-free. The physical examination was essentially negative, except for a moderate degree of hypertension. Blood pressure, 150/90. The scleræ and fundi were normal. At some time during the period of observation the following special examinations were made.

Blood Wassermann, negative; basal metabolic rate, −14 per cent. X-ray of the lungs, normal; x-ray of legs—the arteries not visualized. Blood count—red blood cells, 4,500,000; white blood cells, 9,000. Differential count was normal. Blood chemistry—sugar, 131 mg. per cent; urea, 23.3 mg. per cent; uric acid, 3.02 mg. per cent; cholesterol, 183 mg. per cent.

During the months which followed it was repeatedly observed that twenty-four hour specimens of urine always contained large amounts of sugar, whereas the blood sugar was usually within normal limits, and even at times reached hypoglycæmic levels, although our patient never gave evidence of having experienced symptoms associated with hypoglycæmia.

Because of these observations a glucose-tolerance test was made, with results consistent with a diagnosis of diabetes mellitus (Chart 1). This still left to be explained the continued and pronounced glycosuria present in this patient with normal and subnormal blood sugars. We were led to postulate a lowered threshold of sugar associated with diabetes mellitus. To test this hypothesis the following experiment was made, with the view of determining the "descending" and "ascending" threshold for glucose.



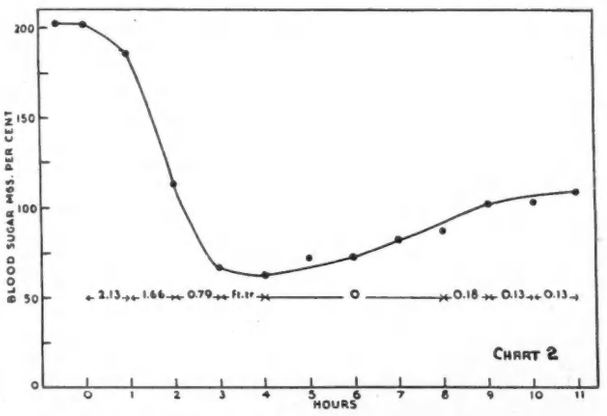


Chart 1.—Glucose time curve after 100 grams of glucose. Blood sugars for this and subsequent determinations by Folin-Wu technique. **Chart 2.**—First arrow—level of blood sugar fasting. Second arrow—meal, 40 units of insulin hypodermically. The figures between darts show the total glucose excretion in grams in the urine in the period indicated. The cholesterol for the fasting specimen, 175 mg. per cent, CO_2 combining power 58.9 volumes per cent.

* From the Metabolism Service, Jewish General Hospital, Montreal.

The evening before the experiment the patient received her usual meal, but with insulin omitted. At 8 a.m., while she was fasting, a sample of blood was drawn on which the sugar content, carbon dioxide combining power, and cholesterol were determined. Her bladder was emptied, and after a qualitative test for sugar in the urine had been made the specimen was discarded. Her breakfast was then ordered, consisting of protein, 25 g.; fat, 20 g.; carbohydrate, 75 g.;* and 40 units of insulin hypodermically were given just before the meal. Blood and urine were collected at hourly intervals for quantitative glucose determinations. In addition, the reducing substance in the first sample of urine was tested qualitatively, and found to be fermentable and to give a negative test for pentose.

The data from these analyses are recorded graphically in Chart 2, from which it will be observed that from the fasting level the blood sugar fell within three hours to a hypoglycæmic level, and at the fifth hour began to rise, reaching a normal fasting level by the seventh hour. During the period of falling blood sugar sugar appeared in the urine in the quantities written on the chart, until the blood sugar had reached a level of 65 mg. per cent. After this the urine remained sugar-free until the blood sugar reached 101 mg. per cent, when glycosuria reappeared and persisted until the conclusion of the observations.

It would appear that this patient has a threshold for sugar which lies between 113 and 65 mg. per cent, as the blood sugar falls, and between 86 and 101 mg. per cent as the blood sugar rises.

The fact is noteworthy that during the fairly long period of hypoglycæmia she experienced no discomfort.

DISCUSSION

The importance of establishing the diagnosis in such a case as outlined is not without practical significance. Glycosuria obviously offers no guide to the degree of glycæmia, so that frequent blood-sugar determinations are essential to establish the degree of control of the diabetes. The energy requirements of a patient already restricted by the necessarily limited food intake are still further depleted by loss of carbohydrate in the urine. For this reason the doses of insulin must be such as to reduce the amount of carbohydrate lost in the urine to a minimum. This is accomplished by keeping the blood sugar at the lower limit of the normal. In our particular patient this was obtained by a diet of 1,500 calories and insulin 15-0-15.

The sequence of the development of the two conditions is not known in this case. The renal glycosuria, however, is probably a congenital defect. It may be assumed that the tolerance to low levels of blood sugar in this case represents a protective physiological adjustment.

* Through misinterpretation she was actually given one-third of this amount.

SUMMARY

A case of renal glycosuria associated with diabetes mellitus, with the diagnostic criteria, is reported.

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PUERPERAL INFECTION*

BY ROSS MITCHELL, M.D.

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IF there is one morbid condition that may fitly be called "the disease of women", it is puerperal infection, a disease which strikes at the very roots of life. From the beginning of man's existence on this planet it has threatened the reproduction of the human race, and though there no longer exist those great epidemics which raged in the hospitals of Europe in the seventeenth and eighteenth centuries, puerperal infection still retains its priority among the causes of maternal deaths. It is the arch-foe of motherhood.

HISTORICAL

The disease was well known to Hippocrates, who described a fatal case occurring in the wife of Philinus. Galen and Avicenna also wrote descriptions. The first to point out the contagious nature of the disease were Charles White, of Manchester, (1773) and Alexander Gordon, of Aberdeen (1795). In 1843 Oliver Wendell Holmes, then the young Professor of Anatomy and Physiology at Harvard, published his classical essay, "The Contagiousness of

* Read before the Canadian Medical Association, Alberta Division, Calgary, September 17, 1936, and the Saskatchewan Medical Association, Saskatoon, September 24, 1936.

Puerperal Fever'' with its heart-stirring passages. It is, however, to Philip Ignaz Semmelweiss (1818-1865) that the honour is due of definitely establishing in the minds of men that puerperal fever is due to contagion introduced from without. Beset with opposition by his superiors in rank and thwarted in his just claims to advancement in academic position, his sensitive nature gave way and he died in an asylum, but not before he had revealed the truth. The criticism of jealous rivals could not prevail before his obvious success in reducing maternal mortality by insisting on the cleansing of examining hands with chlorinated lime water. The next great advance came when Pasteur showed that the infective agent was a germ, and that germ the streptococcus. A little later, Lister, by excluding organisms from gaining access to wounds, established modern surgical methods and revolutionized surgery.

The reproach has long been laid against obstetrics that it has not been able to duplicate the success of surgery in preventing infection, but the reproach is not wholly deserved, since the problem of preventing septic infection is a more difficult one in obstetrics than in surgery. The facts that labour usually lasts several hours, that it entails severe muscular exertion, bruising of tissues, and loss of blood, that the uterine cavity communicates both with the outside of the body and with the peritoneal cavity, and that the perineal and the perianal skin cannot be disinfected, show how readily infection may arise in the parturient woman.

The Increasing Hospitalization of Maternity Patients

A change in the practice of obstetrics in recent years has some bearing on the problem of infection. This may not be so evident in rural districts, but in cities the change within the last twenty-five years has been marked. During the year 1912, in Winnipeg, 31.5 per cent of registered births occurred in hospitals and maternity homes, and during 1935 the percentage had risen to 92. That means increased risk of infection, since the parturient in an institution has more contacts and is exposed to more organisms. However if proper precautions are observed in an institution the incidence of infection may be no greater, or even less, than in the home.

Bacteriology

Pasteur found the streptococcus to be the chief infective agent in fatal cases, and that finding still holds good today. Munro Kerr, in his recent work, ''Maternal Mortality and Morbidity''[1] states:—

> ''As far as can be judged at present, the position would appear to be approximately as follows: (1) Fatal cases—*Streptococcus hæmolyticus* is responsible for 75 to 85 per cent; anaerobic streptococci are responsible for about 5 to 10 per cent; pneumococci, *Staphylococcus aureus, B. coli, B. Welchii*, etc., are responsible for 5 to 10 per cent. (2) Grave cases—here again *Streptococcus hæmolyticus* is the chief infecting organism, but anaerobic streptococci are the cause in a higher proportion in this group than in the fatal cases; the same applies to other infecting organisms. (3) Mild cases—anaerobic streptococci and any of the other organisms mentioned are the organisms present in this group; *Streptococcus hæmolyticus* is very infrequently the infecting organism.''
>
> The recent work of Lancefield, Hare and Colebrook has shown that it is possible to divide the hæmolytic streptococci into various serological groups, A to G, of which Group A is responsible for the serious puerperal infections. Lancefield and Hare[2] showed that the hæmolytic streptococci found in the vagina before labour do not belong to Group A. A subsequent paper by Hare and Maxted[3] disproved the possibility of the bowel as a source of infection from Group A hæmolytic streptococci. Colebrook, Maxted and Johns[4] also failed to find Group A streptococci on the perineal and perianal skin of any of 150 women, though they found them in 181 normal individuals on the skin of the hands, where their presence was attributed to contamination from the mouth. The methods of Griffith and Lancefield, *viz.:* agglutination and precipitin reactions have made it possible to prove or disprove the identity of streptococci obtained from different sources. Thus while two or more contacts may harbour hæmolytic streptococci, it is sometimes possible to prove that the organism recovered from one contact is identical in every respect with that recovered from the patient, while the organism from the second contact may be quite dissimilar.

The Sources of Infection

The late J. O. Polak said there were eleven sources of infection, ten fingers and the nasopharynx. Though these are not the only sources, they are undoubtedly the outstanding ones. Infection may arise before labour, during labour, or in the puerperium. To prevent infection through fingers, vaginal examinations should be kept to the minimum, and when performed should be conducted with the same strict technique as the conduct of labour itself. The technique of the Winnipeg General Hospital is as follows. The hands and forearms are scrubbed with running water, soap, and a sterile brush, the nails cleaned with nail file or orangewood stick, and the scrubbing repeated, using a second brush. The total scrubbing lasts seven minutes. The hands and forearms are then washed in alcohol, and a

sterile cap, gown, mask and gloves are put on. From time to time the gloved hands are rinsed in a solution of lysol, 1/200. To prevent infection from the patient, the vulva and lower abdomen are shaved, and these parts, together with the inner side of the thighs to the knee and the perinæum, are washed with soap and water, then sponged with lysol, 1/200, or painted with alcohol and acetone solution of mercurochrome. Particular attention is paid to cleansing the folds of the prepuce of the clitoris. Finally the patient is draped with sterile sheets, leggings, and towels.

Infection from the nasopharynx is prevented by masking the nose and mouth. A four-ply mask of fine meshed gauze, according to Meleny, prevents organisms from being blown, or sneezed, or coughed through it. Any person with an acute infection of the nose or throat is a danger to the parturient woman. The human respiratory passages constitute the only important stronghold of those strains of hæmolytic streptococci which are capable of initiating severe puerperal infection.[5] Masks should be worn by attendants during the whole course of labour, and by nurses giving perineal care during the puerperium. The throats and noses of nurses, interns, and attending staff should be swabbed for hæmolytic streptococci before they begin their term of service on maternity wards, and the swabbing should be repeated at least once a month. Carriers of hæmolytic streptococci should be kept from attendance on maternity patients until swabs of the nose and throat are negative. This applies also to those with acute respiratory infections of any kind.

There is a seasonal incidence of hæmolytic streptococci puerperal infection corresponding to the seasonal incidence of sore throat and other streptococcal disease, cases being most prevalent in the months from January to May. If the patient has a sore throat or cough on admission she should wear a mask during labour, when nursing her baby, or when perineal care is being given.

Other sources of infection are: (*a*) the patient herself, either from self-examination, the presence of a streptococcal lesion on or within her body, or exposure to infection from members of her family before labour has set in; (*b*) marital intercourse shortly before labour; (*c*) instruments, dressings or bedpans.

PATHOLOGICAL ANATOMY

In the vast majority of cases the primary source of puerperal infection is the placental site, which offers an ideal culture medium for the growth of organisms. The most common lesion in puerperal infection, then, is septic endometritis, which may be limited to the placental site or involve the whole lining of the uterus. When the less virulent organisms are the infective agents, the infection is usually limited to the uterus but when the hæmolytic streptococcus invades the uterus the organisms make their way through the lymphatics from the interior of the uterus to the peritoneal surface and there give rise to peritonitis which may be either local or general. Parametritis, *i.e.*, inflammation of the periuterine connective tissue, may follow infected tears of cervix, or may be due to spread of infection from a septic endometritis via the lymphatics. The inflammation may be mild, when the uterus feels as if it were set in plaster, or an abscess may form. Infection may also be carried in the blood stream from septic thrombi at the placental site giving rise to thrombophlebitis in the pelvic saphenous veins, or to pyæmia with the formation of metastatic abscesses in any part of the body.

In 89 autopsies Kneise found the following lesions:

	Cases
Peritonitis	43
Thrombophlebitis	20
Pyæmia	17
Parametritis	7
Sepsis foudroyante	2
	89

PROPHYLAXIS

Since puerperal infection has such possibilities of death or invalidism, and since there is no specific cure, prophylaxis is all important. A recent report of the Medical Research Council of Great Britain by Dora C. Colebrook[5] contains important practical conclusions drawn from a study of cases of puerperal infection admitted to the Isolation Block of Queen Charlotte's Hospital. She points out (1) the importance of removing a patient from a heavily infected environment, whether the extra-genital strains are "familial" or "professional"; (2) the urgent need for the education and assistance of the patient in the practice of scrupulous

personal cleanliness, of the disinfection of her hands, and even, on occasion, of the use of a mask; (3) the desirability of making a bacteriological examination of a swab from the uterus of every febrile puerperal patient, and a search for carrier strains when the diagnosis of hæmolytic streptococcal infection is established. The finding of hæmolytic streptococci in the uterus, she says, will point to the potential seriousness of the infection, and the discovery of an extragenital strain will lead to the adoption of adequate measures for the protection of other patients. In the Winnipeg General Hospital any puerperal patient with a rise of temperature to 100.4° F. on any two successive days, except the first, is isolated. Recently a complete isolation block has been created in that hospital for infected mothers and their babies. Dora Colebrook concludes her report thus: "Clinical experience, then, lends support to the experimental evidence in showing that by the use of scrupulous care, an intelligently devised antiseptic and adequate masking, the risk of infection may be successfully overcome."

TREATMENT

The viewpoint of the authorities of the previous generation is expressed in the dictum of C. M. Green: "Virulent infection yields to no treatment save the grace of God." Today, such a resigned attitude is less justified, for there is good reason to believe that in some cases patients' resisting power may be raised and the infection overcome, either by natural or acquired immunity. Natural immunity may be obtained by removing the patient from a heavily infected environment and supplying fresh air, sunlight, good nursing, simple nourishing food, forced fluids, sleep, and freedom from worry. If possible, the patient should be in the open air for at least a part of the day. Adequate drainage of the uterine cavity is important, and may be most easily secured through the Fowler position in a Gatch bed. The late Remington Hobbs advocated the instillation of sterile glycerine into the uterine cavity through a catheter. He claimed that the hygroscopic properties of glycerine promote a flow of lymph from the uterine walls to the cavity, thus preventing the spread of infection through the walls. Any method of intrauterine manipulation, however, has possibilities of harm, and if this method is employed it should be with the utmost gentleness.

Various antiseptic substances, such as colloidal silver, colloidal iodine, acriflavine, mercurochrome, metaphen, and charcoal have been injected into the blood stream in the hope of killing the invading organism without injuring the host, but in spite of occasional successes chemotherapy has been disappointing. When the organisms are sealed away in septic thrombi from the circulating blood the task set the antiseptic, to search out and destroy these organisms, seems impossible.

Various attempts have been made to create artificial immunity. When the causative agent breaks down red blood cells, as does the *S. hæmolyticus,* it seems reasonable to supply fresh blood from a compatible healthy donor. If blood transfusions are given they should be small in quantity and repeated at intervals of a few days. Massive transfusions have been shown to be actually harmful. J. O. Polak was a strong advocate of repeated small whole blood transfusions. Cadham,[6] however, claims that even better results are obtained with blood serum. Sixty to one hundred c.c. of blood are withdrawn from a suitable donor, allowed to clot at room temperature, and subsequently placed in an ice chest for 12 or 18 hours. The serum is then removed and the patient transfused with it. In this way the patient's complement is raised. In addition to human serum, injected on alternate days, he seeks to build up an artificial immunity by injecting serum from immunized rabbits. The procedure is as follows. When a positive blood culture is obtained from a patient showing symptoms of septicæmia, a vaccine of the microorganisms is prepared as rapidly as possible (generally within 24 hours) and this vaccine is inoculated into a series of rabbits immunized against numerous strains of streptococci and staphylococci. On the following day, from 6 to 10 c.c. of blood are withdrawn by means of a vacuum tube from the heart of one of these rabbits, the blood is allowed to clot, the serum is removed, tested for sterility, and the patient is then inoculated subcutaneously or intramuscularly with this serum. Inoculations with the vaccine of the causative organisms are continued daily with the series of rabbits already immunized and which have been selected for use in the

particular case under treatment. If necessary, the serum from the blood of these rabbits in rotation is administered to the patient on successive days. It is highly important that the serum be given as early in the disease as possible. Up to February, 1936, Cadham had treated 41 patients severely ill with puerperal septicæmia with 36 recoveries, a highly encouraging result, since the mortality rate in puerperal patients with positive blood cultures is estimated at 85 per cent.[7]

Operative treatment is occasionally indicated. Parametritic abscesses should be opened as soon as fluctuation appears. In cases of peritonitis good results have been obtained from laparotomy when the infection is due to the gonococcus, but almost never when due to the streptococcus. Ligation or excision of the pelvic veins distal to the thrombus, as is done in the case of infected thrombi complicating mastoid disease, is sometimes a life-saving operation, but the great difficulty lies in deciding when it should be performed in the individual patient.

CONCLUSION

It would appear, in the light of present knowledge, that the best means of safeguarding the pregnant woman from puerperal infection are: to supervise her environment, especially during seasons when respiratory infections are common; to educate her in habits of scrupulous personal cleanliness; to conduct labour with the care and painstaking technique associated with surgical operations, including especially masking of the nose and throat; to prevent as far as possible tissue trauma and loss of blood; to see that proper care is taken in performing the toilet of the perineum; and to isolate any patient who shows evidence of puerperal infection. Only through the conscientious carrying out of these measures can there be any hope of reducing the mortality and morbidity arising from puerperal infection.

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A NEW METHOD OF RHINOPLASTY FOR SINKING OF THE TIP OF THE NOSE*

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ALL surgeons who devote themselves to the æsthetics of the nose admit that it is very difficult to correct a sinking of the tip of the nose, and by intervention to obtain perfect results. The methods described do offer a certain improvement but, generally speaking, do not satisfy the interested parties. Last September I operated on a patient who presented this type of nasal deformity, and, having recourse to a new process, I succeeded in pleasing him.

CASE REPORT

L.S., a male, 22 years of age, sought my advice on September 23, 1935, about a depression of the end of the nose. He said that he never received any traumatism, and that his deformity was congenital.

On examination I observed that the tip of the nose was particularly soft and much sunken; the nostrils were slightly dilated; the nasal bones were normal. In short, the malformation was solely localized to the inferior region of the cartilaginous septum. Anterior rhinoscopy revealed a well defined rectilinear septum, without thickening. The turbinated bones had not increased in size. There was nothing interesting to note in the rhino-pharynx or in the ears. To deal with this situation I proposed a rhinoplasty by means of costal cartilage, and, with the patient's consent, the operation was fixed for two days later. An antiseptic nasal salve was then prescribed. Half an hour before the operation, I injected a solution of H.M.C. (hyoscine, morphine, cactoid).

Operation.—Having cut the hairy follicles, irrigated and sterilized the nasal fossæ, I gave a local injection of novocaine-adrenalin. Under the control of the index finger placed in the right nostril, I incised to the left, on all its length, the mucous membrane which separates the septum from the columella. Great care was exercised in order to avoid any perforation to the right side. Having dissected all the neighbouring tissue of this sectioned membrane, to render it more elastic, I dug a tunnel over the tip of the nose to the nasal bones. Always under local anæsthesia, I took a long piece of cartilage from the first floating rib on the right side. Stripped of its perichondrium, it was separated into two, and the thinnest part, after being modelled, was

* Read before the "Premier Congrès Européen de Chirurgie Structive", Bruxelles, October 3 and 4, 1936.

placed in the nasal vestibular wound. This graft, in direct contact with the quadrangular cartilage, took its point of support from the superior maxilla, pushing up the tip of the nose. Silk and very thin needles were used to carefully suture the wound. In order to increase the æsthetic effect and at the same time to protect the tip of the nose against pressure caused by the extremity of the cartilaginous insert, I made a small external vertical incision on the skin which covers the columella, with the object of rejoining the tunnel already dug. This communication established, the second piece of cartilage, the thicker, was then cut in two lengthwise, and after modelling one part was introduced by this opening as far as the nasal bones. Finally, the second half was placed at the tip of the nose in order to cover the extremity of the graft. I then sutured the cutaneous incision with silk by means of small conjunctival needles, and a light compressive dressing of the two nostrils was adjusted. This wound was protected with a piece of iodoform gauze maintained by two bands of adhesive plaster. According to my habit in my cases of rhinoplasty, I applied finally, under the pyramid, a splint of copper, retained again this time by adhesive tape. Three layers of sutures closed the costal wound, and I finished the operation with the ordinary abdominal dressing.

Post-operative sequelæ were most simple, and the dressing of the nostrils, practised after two days, was then renewed every twenty-four hours. From the seventh day I began to remove the stitches of the skin covering the columella, and those of the vestibule. I waited a little later before removing the sutures of the costal wound. Sixteen days after the intervention the patient left the hospital absolutely cured. By this time the tip of the nose was well remounted, and was to be found in direct line with the nasal bones. The nostrils had a normal opening. Eight months after the operation, the patient wrote me that he was quite satisfied, and that his nose was perfectly straight.

In appreciating the different methods available for correcting a sinking of the tip of the nose, we see that each malformation is an individual problem. In the case of a nose too long and slightly flattened at its point we must, by the endo-nasal route, separate the columella from the septum, resect small slices of the quadrangular cartilage, separate the skin of the nasal pyramid, and suture on each side the vestibular mucous membrane, after having removed the exuberant part. In order to definitively stabilize this skin and at the same procedure lift up the tip of the nose we must apply bands of adhesive plaster with tractions at first vertical and afterwards lateral.

If the sinking manifests itself on a soft nose, with nostrils sufficiently large, Joseph, of Berlin, advises the entire detachment of the columella from the septum by means of an external incision, to uncover the two alar cartilages, and then to suture them in order to bring them nearer and to lift up the tip of the nose. In certain cases this method can give fair enough results, but generally the cicatricial tissue which forms consecutively to the intervention has the unfortunate property of damaging with time the æsthetic appearance. The operation can also be performed by the endo-nasal route, but it is easy to conceive that this one is much more laborious and more problematical as to its results.

Sheehan, of New York, advocates an incision of the skin which covers the columella, and the digging of a tunnel on the tip of the nose. Having taken a piece of costal cartilage, he models it in such a way that two parts, about equal and bent at a right angle, are retained by the perichondrium. This graft is then placed at the tip of the nose as far as the nasal bones and in the wound of the columella. In hands as skilful and experienced as those of the originator of this method one may expect excellent results. However, we must admit that for obtaining a good correction the graft must be perfectly prepared from all points of view, and that the mucous membrane of the nasal vestibule on each side must not be damaged.

Moulonguet, of Paris, in the case of a patient who came to consult him for a very marked sinking of the tip of the nose, consecutive to a traumatism dating back to early infancy, where there was no cartilage of the septum and of the columella, intervened in the following manner. Under anæsthesia with chloroform, a small transverse incision of 0.5 cm. in length was made just below the end of the nose, and a subcutaneous canal of 4 cm. was dug as far as the nasal bones. Then a second incision was made starting from the middle part of the first one transversely, and bisecting the columella and the septum to a depth of 1.5 cm. He then took a piece of cartilage from the edge of the ninth rib stripped of its perichondrium, presenting more or less the form of a square, the longest branch of which was 2.5 cm. This graft, once placed, had its point in contact with the nasal bones, its inferior part, with the nasal spine, and its superior portion pushing up the tip of the nose. The author of this process reports having obtained a very good æsthetic result as to profile. As to the front view, the graft took a slight curving to the right, which forced him later to have recourse to an injection of paraffin to correct this. If now I understand this method, I must suppose that the patient of Dr. Moulonguet had desired him to practise the intervention under chloroform. Indeed, it would have been much easier to perform it, even without any pain, under local anæsthesia, as this would have allowed him to distend the tissues

up to a certain point, and especially those of the nasal septum, which, according to his description, were reduced to "a simple mucous fibre". I do not understand the need of a transverse incision, 0.5 cm. in length, below the tip of the nose, before digging a tunnel as far as the nasal bones, nor the necessity for making a subcutaneous canal of 4 cm. as far as these same bones, when it was necessary to put in a cartilaginous graft of but 2.5 cm. In this particular case the result was very good. However, I doubt whether this method could be applied practically for the correction of a sunken tip of the nose in general, because, even if adapted to the circumstances, there would always be the danger of a perforation of the mucous membrane of the nose at the time of the bisection of the columella and of the septum. We all know that this little accident might have as a consequence an infection at first, and then the elimination of the graft. I shall not criticize the cutaneous cicatrix, which with time almost entirely disappears.

The setting of a cartilaginous stay, to push up the tip of the nose, has already been described, but this simple method is insufficient to give to the pyramid a straight line. Moreover it exposes the end of the nose to become more or less nippled at the place where the pressure is found. The creation, from the front to the rear, of a tunnel in the columella, after an incision from 5 mm., to 1 cm., recommended by certain authors, is not practical, because this tunnel is too small to receive a graft strong enough to definitely push forward the tip of the nose, and, besides, its application brings danger of rupture during laborious manœuvres. With the piece of cartilage which I introduce by the external route as far as the nasal bones I can obtain at will the desired correction. Finally, the last graft, placed in contact with the preceding one, has for its object not only to increase the effect of the nasal correction but also to protect the tissues in covering the cartilaginous beam at its lobular extremity.

It is very important to place the index finger in the right nostril during the section, from the front to the rear, of the mucous membrane, which separates the columella from the septum to the left, so as to exercise a control and consequently prevent perforation. In order to give more extension to this mucous membrane incised, with the object of introducing a cartilaginous stay as large as possible, absolutely straight and free from its perichondrium, we must have recourse to a dissection of its borders. The graft is then placed in contact with the septum, and it is easy to perfectly protect it in suturing with silk, by means of very fine needles, the vestibular wound. Finally, there remains but to practise a small vertical incision below the tip of the nose, to connect the opening with the tunnel already created at the time of the section of the mucous membrane separating the septum from the columella, and to place therein the two pieces of cartilage. Suture of this cutaneous incision, light compressive dressing of the two nostrils with iodoform gauze, external dressing, and application on the nasal pyramid of a splint of copper retained by bands of adhesive plaster, and ordinary closing of the costal wound complete the operation.

For all rhinoplasties dealing with a pronounced lesion where it is necessary to have recourse to a graft I employ the cartilage, (as I have already stated in a previous paper) and, in preference, the cartilage of the first floating rib.[1] Indeed, the anatomy of this costal extremity adapts itself particularly well to the modelling, and this graft alone is amply large for the correction of all deformities produced by an insufficiency of the nasal skeleton. Moreover, this cartilage can be taken without pain, under local anæsthesia, and the post-operative sequelæ are always very simple.

In the case of a nose flattened at its end, widened and stretched, we must at first bring nearer the alæ, according to the process of displacement well described by the authors, before lifting up the tip of the nose with a cartilaginous beam, and then applying, by the external route, the two other grafts.

I believe I have sufficiently described in this paper the method that I have devised for the correction of the sinking of the tip of the nose, and emphasized its superiority over all others. This operation is particularly easy, is practised without any pain under local anæsthesia, and assures the best possible guarantees as to the æsthetic results.

I regret to be unable to furnish any illustration, because my patient refused to be photographed either before or after the operation.

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HÆMATOMA OF THE ABDOMINAL WALL SIMULATING INTRA-ABDOMINAL TUMOUR

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INTRAMURAL lesions of the anterior abdominal wall are rarely mentioned in the differential diagnosis of various intra-abdominal lesions, and in some textbooks no reference is made to this condition. Until one has had an actual experience and has searched the literature for reports it is surprising that many careful observers have failed to recognize a condition which may simulate several abdominal entities.

The rectus abdominis muscle is enveloped anteriorly and posteriorly by the aponeurosis of the internal oblique and transversalis muscles, but the semilunar fold of Douglas marks the lower limit of the posterior sheath, and below this point only the aponeurosis of the transversalis and peritoneum separate the muscle from the peritoneal cavity. Between a point opposite the umbilicus and the insertion of the muscle over the 5th, 6th and 7th ribs there are three lineæ transversæ equi-distant, extending nearly through the muscle and dividing the latter into compartments. The lowest fibrous intersection is opposite the umbilicus and the highest above the level of the xiphoid. The deep epigastric artery sinks into the substance of the rectus after it passes over the semilunar fold of Douglas, which is at the level of the navel.

Painful sensations in the abdomen may arise from various extra-abdominal sources, and in approaching a case where pain is referred to the abdomen and associated with a swelling there it is important to keep in mind the possibility of an intramural lesion of the abdominal wall. Diagnostic errors are more often those of omission than commission, and in the investigation of abdominal catastrophies an unusual condition may be overlooked by the observer whose thoughts automatically centre upon the obvious clinical diagnosis suggested by certain symptoms and findings, not realizing, much to his chagrin, the possibility of the unlooked-for lesion. A rude awakening to the true nature of the disease causes one to ponder and ask "Why was that not considered in the differential diagnosis?" The answer is simple —the existence of such a clinical condition had not suggested itself to the observer.

CASE REPORT

A woman, 75 years of age, was getting out of the left side of her bed about noon, July 27, 1926, and was suddenly seized with a severe pain in the left side of the abdomen. When seen shortly afterwards she appeared slightly pale, was suffering considerable pain; her temperature was normal, with little disturbance in the pulse rate. Upon inspection of the abdomen no abnormality could be seen. There was some rigidity, more marked on the left side, and one could feel an elongated, vertically situated swelling on the left side, about 3 inches long and 2½ inches broad. Its lower margin was about opposite the level of the umbilicus, and one could insinuate the fingers between the ribs and the upper limits of the swelling. It appeared to be smooth, non-fluctuating and was tender, movable laterally, but not up and down. The patient had been in her usual health and there was no history of any previous gastro-intestinal distress. After removal to hospital her sole complaint was of pain upon attempting to move. The liver and spleen were not enlarged. No other masses could be detected, the left costovertebral angle was not occupied by any swelling, and no tenderness could be elicited there.

The condition presented difficulties in diagnosis. But before light was thrown upon the problem one felt that, in the absence of complaints of any previous gastro-intestinal symptoms, something unusual had happened. Accordingly, the aid of x-ray investigations (pyelograms, etc.) was invoked.

A barium enema flowed as far as the cæcum. The sigmoid flexure was rather kinked, and the transverse colon was displaced upwards in the middle portion. No filling defect was detected in the great intestine. A mass, centrally situated, displaced the sigmoid and transverse colon. A barium meal given two days later revealed no pathological condition in the small intestine, but gave evidence of a mass present in the lower left quadrant displacing the coils. Pyelograms of both kidneys negatived a renal neoplasm.

In the meantime, no change took place in the swelling until August 2nd, when the tumour became prominent and ecchymoses appeared in the region of the umbilicus. Under local anæsthesia a syringe with a large-bore needle, introduced through the skin over the most prominent part, withdrew coal black blood, and through a two-inch vertical incision the mass was investigated. The subcutaneous tissues were infiltrated with black clotted blood, and after incising the anterior sheath of the rectus the finger entered a cavity in the muscle, which was torn across, and about 2½ to 3 ounces of blood clot were evacuated. This muscle had been separated from the posterior sheath. No fresh bleeding was present. The torn muscle and anterior sheath were sutured, the wound closed without drainage, and primary healing followed. The patient was discharged from hospital in one week.

It is interesting to note that during the last few years various observers have reported cases of rupture of the rectus abdominis with hæma-

toma, and one is impressed with the difficulties in diagnosis which have been encountered. Cuthbertson collected 41 cases in women in which no fewer than 12 were diagnosed as ovarian cysts and ectopic gestation. In only 3 of the 41 cases was hæmatoma recognized before operation, while in 2 others it was suspected. Of the 41 patients, 13 were over 50 years of age; 7 between 40 and 50 years of age; and 6 under 40 years of age.

Hæmatomata which simulate pelvic growths are below the umbilicus, and are thus due to rupture of muscular branches of the deep epigastric artery. The most interesting thing about these is the diagnosis. The main point is the recognition that these swellings are part and parcel of the abdominal wall. This is generally made by noting that they can still be felt when the recti are in action and that they become fixed as the muscles contract. Another point is that when a mass in the abdominal wall is not too large a resonant note may be obtained through it by deep percussion. But these signs are not infallible, for a mass within the abdomen that has become adherent to the wall gives the same signs as one which is a part of the wall. Even physical signs can tell lies and must be checked.

Halperin, in a review of the literature, remarks that this apparently rare condition was known to physicians of antiquity. Thus Maydl,[1] in his classical contribution entitled "Subcutaneous rupture of muscles, tendons as well as tear-fractures", gives accurate descriptions of the condition as made by Hippocrates and Galen. In his extensive review of the literature covering the period from 1809 to 1880 Maydl found 14 cases of spontaneous rupture of the rectus abdominis muscle.

Of greater interest to surgeons, however, are the cases of spontaneous hæmatomata of the abdominal wall occurring in elderly women without any apparent cause. The contributing factor in these cases may be so insignificant as to escape notice. Commonly, they are the result of a cough or coughing spell. There is usually an abrupt onset with severe pain in the abdomen, associated with signs of peritoneal irritation, such as belching, nausea, vomiting, obstipation, abdominal distension and muscle rigidity. A swelling which is painful on palpation develops either to the right or to the left of the median line. Diagnostic errors here are all due to the fact that it is believed that an acute intra-abdominal condition exists. The most frequent diagnosis is that of a twisted ovarian cyst.

The condition has been mistaken for ovarian tumour, acute appendicitis, ileus, incarcerated hernia, and cholelithiasis. The diagnosis of an acute intra-abdominal process is particularly suggestive in cases in which the hæmatoma develops in the lower third of the muscle below the semicircular line of Douglas. The absence at this level of the aponeurotic sheath on the posterior surface of the muscle permits the extravasated blood to bulge into the peritoneal cavity.

In Wohlgemuth's series 79 took place below the navel, and in 18 the rupture was below the semicircular line of Douglas. It is also worthy of notice that the muscle below the navel is narrower and has fewer or no tendinous bands. In 7 cases of spontaneous hæmatoma collected by Hilgenreiner up to 1924 not one was correctly diagnosed before the operation.

Although the intramural existence of the lesion be recognized, the true condition may still be confused with an incarcerated hernia, a tumour of the abdominal wall, such as a sarcoma, desmoid, fibroma, or with gumma of the rectus muscle, or tuberculosis or actinomycosis of the abdominal wall.

Bloody discoloration of the skin may develop over the swelling or about the navel, with streaks running towards the symphysis pubis. The pathogenesis of this group is not quite clear. The question as to whether the muscle ruptures first or whether the rupture of the epigastric artery takes place primarily, with a secondary loosening and tearing of muscle fibres, is debatable. In the absence of a violent effort, degenerative changes in the muscle are responsible. Such cases have been observed in certain infectious diseases, such as tetanus, typhus fever, miliary tuberculosis. In the spontaneous hæmatomata of elderly women obesity, a pendulous abdomen and atheroma of the blood vessels undoubtedly play an important part.

Halperin's case is very much to the point.[6]

A woman of 75 years, turning from one side to the other at 4 a.m., February 5, 1924, felt a stabbing pain in the upper right abdomen, more severe than previous stomach attacks, which were attended by epigastric distress and tenderness, and diagnosed as gallstone disease. At 6 a.m. there was a tender hard mass extending from

the gall-bladder area slightly below the level of the umbilicus, 3 inches in diameter and with well demarcated edges. On February 6th her symptoms had abated slightly. X-ray examination that day added nothing except a misleading suggestion that a tumour mass was pressing upon the abdomen from behind. On February 7th ecchymoses appeared in the skin about the umbilicus and right abdominal wall. On February 8th right rectus incision was made. The sheath of the rectus was incised and a large quantity of clotted blood was removed from the cavity within the sheath. The peritoneum was intact and was not opened. There was no active bleeding and no fresh blood. In the bed of the cavity containing the clotted blood was the distal portion of the deep epigastric artery, dissected free from surrounding tissue for a distance of two cm. Its anastomosis with the superior epigastric artery could not be demonstrated and had probably been destroyed.

The writer's case presented difficulties in diagnosis and the solution became apparent only when the mass was visibly prominent and ecchymoses appeared. Upon the true condition being suspected, it was found that by grasping the mass laterally with both hands and attempting to draw it away from the abdomen the association of the swelling with the parietes was revealed, as the patient's abdomen was somewhat pendulous and muscles soft and relaxed.

SUMMARY

1. Rupture of the rectus abdominis muscle with the formation of a hæmatoma is rare.

2. Various tumours of the anterior abdominal wall, such as sarcoma, desmoid, incarcerated hernia, etc., may simulate intra-abdominal new growths.

3. An abdominal swelling with pain may be due to an intramural lesion, especially when occurring below the level of the umbilicus.

4. The condition most commonly occurs in elderly people, where changes in the muscles and arteries are present.

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A TABLE FOR THE DEGREE OF INVOLVEMENT IN CHRONIC ARTHRITIS*

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CHRONIC arthritis (or "rheumatism") continues to be a complex and controversial subject, as evidenced by the numerous conflicting reports in the literature. Much of this confusion may be due to the failure of many authors to give sufficient attention to the numerous variations that occur during the course of the arthritic process. Comparable to other chronic diseases, like tuberculosis and syphilis, most types of arthritis pass through various stages of severity, ranging from mild to extreme. The joints may show little or no actual damage, or they may be partially or completely destroyed. Accordingly, the arthritis patient may vary from the early ambulant state to that of the helpless bed-ridden cripple. Therefore, in evaluating reports dealing with "chronic arthritis", and particularly those concerning treatment or "cures", it would seem necessary to take into account these wide variations in the clinical picture of this disease group.

The situation may be improved somewhat by the adoption of a standard "table of degree", to indicate the severity and extent of involvement of the arthritis. The table proposed herein is necessarily descriptive in character, and contains four divisions, or "degrees", varying from minimal to advanced stages. Thus it is somewhat similar to the method of grading used in tuberculosis and in syphilis. The present table is designed primarily for rheumatoid (atrophic) and osteo-arthritis (hypertrophic), but with a few modifications it may be used for practically every type of chronic arthritis regardless of the particular terminology used in the diagnosis or classification. These modifications required for other types of arthritis will be presented in detail at a later date.

* Presented in part in a paper to the Medico-Chirurgical Society of Montreal, on January 22, 1937.

The rating in each case is based upon a combination of roentgenological and clinical findings. The latter term includes the available laboratory data and special examinations. Also, an arbitrary time limit is advisable for grading purposes because of the comparatively rapid variations in the clinical course of certain types of arthritis. Six months' duration has proved satisfactory in the author's experience. With these considerations in mind, the majority of cases of chronic arthritis may be grouped conveniently into one of the four stages of the Table as outlined below.

A TABLE INDICATING THE DEGREE OF INVOLVEMENT IN CHRONIC ARTHRITIS

Degree (or stage)	*X-ray changes*	*Degree (or stage)*	*Clinical findings*
I.	*Soft tissue changes: e.g.,* intra- and extra-capsular swelling. Slight decalcification, local or general. Slight or moderate "lipping".	I.	*Slight symptoms:* pains or aches; discomfort; may be stiffness and swelling; or crepitus. Patient active.
II.	*Joint space is definitely altered.* Loss of cartilage or bone substance. Moderate decalcification. Soft tissue changes are more definite; capsular swelling, and adjacent atrophy. Definite osteophytes or spur formations; local hypercalcification.	II.	*Moderate symptoms:* pains or stiffness; or crepitus. There is definite and prolonged localized limitation of motion. Patient may be relatively active.
III.	*Destruction of joints;* subluxations; deformities; local ankyloses; marked decalcification and soft tissue changes. Marked hypertrophic changes; osteophytes and spurs.	III.	*Severe symptoms:* local and general. Marked deformity or limitation. Patient definitely incapacitated; unable to care completely for himself. May be able to walk with difficulty.
IV.	*Extreme widespread joint changes:* destruction; and bony ankyloses. Extreme hypertrophic changes.	IV.	*Extreme symptoms:* although there may be no actual pain, the patient is helplessly crippled, usually bed-ridden.

First degree arthritis patients are ambulatory, and are usually able to continue at their customary activities. The demonstrable joint changes, apart from soft-tissue swellings, are negligible. This group also includes the "pre-arthritic" patients, and the arthritic patients who may be enjoying a period of remission. The diagnosis at this stage is often difficult. Many of these patients have some type of extra-articular condition only, and are frequently diagnosed incorrectly as "arthritis". The prognosis in the vast majority of this group is quite favourable.

Second degree patients may or may not be able to continue at their usual work, depending upon the site of the joint involvement. However, they are usually ambulatory, and are able to take care of themselves. The diagnosis here is more evident. The prognosis remains reasonably hopeful, providing a proper regimen of therapy can be instituted and maintained. Deformities may thus be prevented.

Third degree patients are so incapacitated by demonstrable joint lesions that they are unfit to work and are partially dependent on others for proper care. They may be able to walk for short distances, but usually are limited to a bed and chair (or crutch) existence. The outlook for this group is serious and the prognosis must be extremely guarded. These patients should have institutional care and training to preserve and regain as much joint function as possible.

Fourth degree patients are crippled to the extent that they are completely incapacitated. Most of them are bed-ridden, although occasionally they may be able to sit up in a chair, with assistance. When the activity of the disease-process subsides there may be little or no pain, and their general health may be satisfactory. Under such conditions some degree of function may be restored to the destroyed joints by suitable reconstructive operative procedures.

The first and second degrees probably include the majority of the "arthritic" or "rheumatic" patients in the out-patient departments of hospital clinics and in private offices. Third degree patients may visit the clinics or offices, but they are essentially bed-cases even though this may be only a temporary phase. Consequently they are seen more frequently in the hospital beds or at home. Fourth degree patients are the unfortunate cripples confined to bed in the homes or in institutions for chronic or incurable diseases, although occasionally they may be admitted to the general hospitals. They are the hapless victims of a

disease process that leaves them with crippling deformities. Incidentally, many if not most of these deformities could be prevented, if the patients could receive suitable treatment in the earlier stages. It is regrettable that there are so few institutions sufficiently endowed and equipped to give them the necessary care.

It is to be expected that the grading of any individual patient may have to be altered from time to time, to correspond with the changes occurring during the course of the arthritis. Variations in the rating between neighbouring divisions are of relatively little import. The significant feature is the separation of the early or mild cases from those in the advanced stage. The inevitable exceptions and border-line cases need not detract from the general function or usefulness of the table. In most instances the roentgenological picture and the clinical findings will be found to correspond fairly well for practical purposes.

The x-rays may be accepted as the standard for grading, because of the greater variability of the clinical features. However, where the x-ray films are not available, or their interpretation proves unsatisfactory, the clinical findings alone must suffice. In patients with polyarthritis, where different joints in the same individual show different degrees of involvement, each joint may be rated separately, if desired, and the final grading is based upon the general average. If however, any one or more of the joints stand out in importance clinically, the rating for the patient should be altered accordingly. Also, where two or more types of chronic arthritis affect the one individual, each type of arthritis should be judged upon its own merits, and graded separately.

In many patients a profound constitutional reaction is found to be entirely out of proportion to the demonstrable physical findings or x-ray changes. Thus it is frequently advisable, for purposes of recording, to have an additional descriptive grading available, *to indicate the severity of the symptoms within each of the four main groups, viz.*, (*a*) mild, (*b*) moderate, (*c*) severe, and (*d*) extreme.

Generally included with chronic arthritis and rheumatic diseases are the large number of cases of extra-articular involvement grouped under the general heading of "fibrositis". There is, of course, no actual joint change demonstrable by roentgenograms, so that the rating in these individuals will have to depend entirely upon the other clinical and laboratory findings. Patients belonging to this group are seldom incapacitated to the extent of requiring a grading beyond 1st or 2nd degree, but they often require the additional ratings to indicate the severity of the symptoms within those groups as mentioned above, *i.e.*, (*a*) mild, to (*d*) extreme.

This whole system of grading arthritic patients has proved of practical value to the author during the past year, particularly for recording purposes. The use of the symbols (I to IV), indicating the degree of involvement, saves time and space in the records, and tends toward a more accurate description of the actual condition of the patient without requiring a long detailed narrative. The same system has been of assistance in x-ray reports, to describe the extent of the individual joint changes.

The Table should prove helpful in survey work for statistical studies. In this connection the clinical findings *dealing chiefly with function* may be used conveniently by non-medical workers, to give a fairly accurate picture of the patients' general condition. The same system may be of assistance in rating patients for disability claims.

It should be obvious that with the wide distribution of arthritic patients and with the many variations in the clinical picture it is difficult to generalize in discussing many features of this disease group. Thus the statement seen or heard so frequently, that a certain number of patients with chronic arthritis have been studied or treated, does not convey a very vivid impression as to the exact nature or condition of these patients. The physical and laboratory findings, the methods and response to treatment, as well as the ultimate prognosis, vary greatly with the type of arthritis and with the stage of development. Observations on patients with 1st or 2nd degree involvement, in most types of arthritis, have a vastly different significance when applied to patients in the 3rd or 4th stages. This is true particularly in reports dealing with treatment. Detailed clinical and laboratory studies are essential in helping to solve the many vast problems of chronic arthritis, and it is hoped that some of these studies may be facilitated by the use of the Table outlined above.

MEASUREMENT OF BLOOD LOSS IN NOSE AND THROAT OPERATIONS

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THE aim of this paper is to report the results of some measurements made to ascertain the actual amount of blood lost in the common operations on the nose and throat. Bleeding during such operations is often troublesome and sometimes serious, especially if post-operative hæmorrhage must be added to that of the operation. Further, our method of control by pressure and packing is still primitive and unreliable, and hæmorrhage remains the chief operative complication in this field.

Clinical experience from the days of blood-letting indicates that a loss of about twenty ounces of blood is the limit of tolerance for the normal adult, and that a greater amount has a definitely unfavourable effect on even healthy persons. The total amount of blood in an adult is estimated at about one-eleventh part of the body weight and somewhat less than that in children and fat subjects. According to Samson Wright a loss of about twelve ounces has no effect on the blood pressure, of twenty-four ounces causes slight but definite depression, and of sixty ounces a very marked fall of blood pressure with "the chances of recovery uncertain". The apparently rapid recovery from a considerable hæmorrhage is somewhat deceptive, and full restoration takes several weeks. In the depressed state of health of most subjects for operation, the bad effects of hæmorrhage appear sooner and are more marked.

Influenced by these considerations I began some years ago to note by means of the suction apparatus the amount of blood lost in some routine operations such as tonsillectomy and mastoidectomy, and in May, 1928, made a brief report of the results at the Winnipeg Medical Society. Since that time the measurements have been continued and the number about doubled. The results obtained prior to 1928 are included in this report as Series 1 and those since then as Series 2. The tabulations show the average of both groups. Many hundreds more measurements were made than appear in the tables, but accuracy within one-half to one ounce is difficult to attain, and when excessive secretion, emesis, or the resort to gauze wipes made a greater error likely the result was discarded. It is probable that such factors as the unavoidable swallowing of blood, a residue adhering to the lumen of the tube, etc., tend to make the result an under- rather than an over-estimation, perhaps to as much as half an ounce in each case. This is close enough, however, for clinical purposes and has provided 903 fairly exact measurements.

In order that the results may be judged some description of the method of operating is necessary. In tonsillectomy and adenectomy the patient lies on the table with the head low and turned to the side, and a self-retaining mouth-gag with a tongue depressor is used. The suction tip has a curved end which serves also as a pillar and palate retractor; it is kept applied below and back of the tonsil, but is not allowed to remain in contact with the tonsillar fossa. Scissors dissection and snare are used for removal of the tonsil, and hæmostats are applied to bleeding points as they appear, and left in place. A sharp curette very lightly applied is used for adenoids, any tags being removed with scissors. The nasal cavities, naso- and hypo-pharynx are cleared with the aspirator and the patient is allowed to leave the table only when the field is dry.

In mastoidectomy the type of operation followed is that which aims at the complete removal of all surgically accessible cells revealed by the radiograph, whether or not they appear diseased. The time required averages about one hour, and the aspirator is used to keep the field clear. In septum resection 20 per cent cocaine in adrenalin, 1/1,000, is applied to the mucous membrane, followed by injection of 1 per cent novocaine. This is done in each case for the purpose of hæmostasis, whether or not a general anæsthetic is used. A special adjustable aspirating speculum has proved very satisfactory for collecting blood. Removal of the middle turbinate is done

by avulsion downward, the posterior end then being cut off with the snare. The operative technique described is so common in its essential features that it might be regarded as standard with the majority of surgeons.

The amount of bleeding which has occurred in these operations is shown in the following Tables.

TABLE I.

BLOOD LOSS IN T. & A. OPERATIONS ON ADULTS (14 YEARS) UNDER GENERAL ANÆSTHESIA

	Average		
	Series 1	*Series 2*	*Total*
Series 1—180 operations Series 2—115 "	7 oz.	5.17 oz.	
Total... 295 operations			6.28 oz.

REMARKS: The maximum loss was 25 oz. The minimum was about 1 oz.; 17 per cent showed a loss of 10 oz. or more. The average blood loss of 8 patients over 50 years was exactly the same.

TABLE II.

BLOOD LOSS IN T. & A. OPERATIONS ON CHILDREN UNDER 14 YEARS UNDER GENERAL ANÆSTHESIA

	Average		
	Series 1	*Series 2*	*Total*
Series 1—133 operations Series 2— 64 "	5 oz.	2.81 oz.	
Total... 197 operations			4.28 oz.

REMARKS: Maximum, 20 oz.; minimum, 1 oz.
12 per cent showed a loss of 10 oz. or more.

TABLE III.

BLOOD LOSS IN T. & A. OPERATIONS ON CHILDREN UNDER 4 YRS. OF AGE UNDER GENERAL ANÆSTHESIA

	Average		
	Series 1	*Series 2*	*Total*
Series 1—15 operations Series 2— 6 "	3.75 oz.	3 oz.	
Total... 21 operations			3.50 oz.

REMARKS: Maximum 12 oz. in first series; 5 oz. in second series. Minimum for both 1½ oz.

TABLE IV.

BLOOD LOSS IN TONSILLECTOMY ONLY ON ADULTS UNDER LOCAL AND GENERAL ANÆSTHESIA

Anæsthetic	*Operations*	*Maximum*	*Minimum*	*Average*
Local.....	27	14 oz.	½ oz.	4 oz.
General...	33	16 oz.	2 oz.	5.2 oz.

TABLE V.

BLOOD LOSS IN MASTOIDECTOMY FOR ACUTE OR CHRONIC MASTOIDITIS

Age Group	*Operations*	*Maximum*	*Minimum*	*Average*
14 yrs. & over	126	30 oz.	1 oz.	7.14 oz.
Under 14 yrs.	64	12 oz.	1 oz.	3.63 oz.

REMARKS: Twenty-six per cent of adults showed a loss of 10 oz. or more. There was no material difference between "acute" and "chronic" or "simple" and "radical".

TABLE VI.

BLOOD LOSS IN SUBMUCOUS RESECTION OF SEPTUM WITH SUPPLEMENTARY TURBINECTOMY. COMBINED LOCAL AND GENERAL ANÆSTHETICS

Age Group	*Operations*	*Maximum*	*Minimum*	*Average*
14 yrs. & over	106			3.50 oz.
Under 14 yrs.	6			6.16 oz.

REMARKS: The minimum is not measurable. The maximum has been as high as 20 oz.; 6 to 7 oz. is fairly common.

TABLE VII.

BLOOD LOSS IN "RADICAL ANTRUM" OPERATIONS (INCLUDING SUPPLEMENTARY SEPTUM RESECTION, ETC.). COMBINED LOCAL AND GENERAL ANÆSTHESIA

Age Group	*Operations*	*Maximum*	*Minimum*	*Average*
14 yrs. & over	49	27 oz.	1½ oz.	11.28 oz.

REMARKS: The high average is noteworthy.

COMMENT

The amounts of blood loss revealed by these measurements will probably be a surprise to most operators. The result of the first series certainly disconcerted me, and the 30 per cent reduction in the second series shows what can be saved by greater caution. While the average amount lost is still higher than many operators estimate it is not a serious matter to most patients. But variations in vascular supply may cause free bleeding in spite of careful operation by accepted technique, and the amount recorded in a considerable percentage of these measurements is large enough to be a factor of some moment in prognosis and treatment.

The table dealing with children under the age of four is worth noting. Such children are known to have proportionately less blood than adults; they are generally fat, and bear loss of blood badly. Their average blood loss of three and one-half ounces after tonsillectomy and adenectomy is, therefore, the equivalent of

several times that amount for the average adult —probably not less than fifteen to twenty ounces. Where the aspirator is used incautiously and no note made of the amount of bleeding so much blood may be unknowingly abstracted that it can be the real explanation of some unfavourable or even fatal results that are otherwise interpreted.

There is no doubt that the aspirator tends to increase the bleeding, both from bone and soft tissues. Emphatically this is true in tonsillectomy if the aspirator is improperly used and applied freely to a bleeding surface in such a way as to aspirate clots and keep the vessels open. The field being thus kept clear,

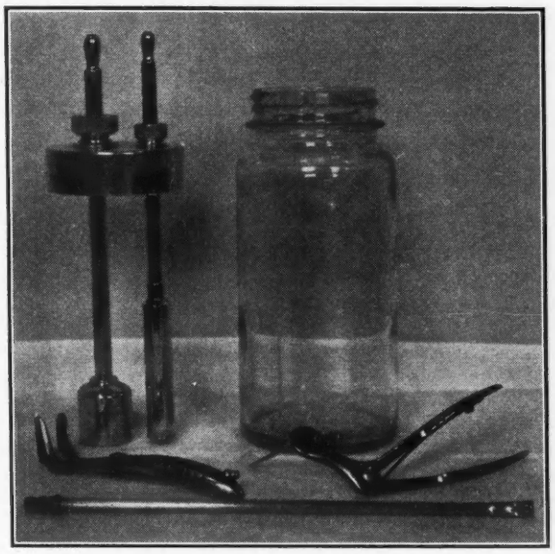

Fig. 1.—Aspirating bottle and suction instruments used in measuring blood loss.

the operator may be quite unaware of the real amount of bleeding unless the blood is trapped in a graduated bottle. In my experience the routine use of such a simple apparatus has been on occasion of great value; for instance, on two occasions of projected double mastoid operation where the blood loss from the first ear was found to exceed twenty ounces, leading to postponement of operation on the second ear. The use of a suction apparatus which sweeps the blood off and down the drain without trace seems unjustifiable. Although the simplest kind of collecting bottle serves quite well, there is some advantage in using one equipped with an adjustable valve which can be set in advance for an amount of bleeding within the margin of safety. The valve closes and stops the suction temporarily at the predetermined level and thus notifies the operator that the set amount has been reached. An illustration of such a bottle with valve which I have had made is shown in Fig. 1.

Two other matters of allied interest may be mentioned. Post-operative hæmorrhage has been practically impossible to measure or even to approximate closely. Nevertheless, nurses should be trained to estimate the amount as accurately as possible, and if the hæmorrhage occurs within a few hours of operation the two bleedings should be regarded as one, and the total amount lost should govern the treatment.

The relationship of the coagulation time of the blood to the amount of blood lost was worked out in a small series. Previous investigations have always dealt with its relationship to post-operative hæmorrhage and no constant relationship to the latter condition has been shown. So far as it was followed, the results indicated that patients with a greater than average operative blood loss showed a slightly lengthened pre-operative coagulation time but the difference was so small that it had no clinical value.

CONCLUSIONS

In conclusion, it would seem that bleeding during nose, throat and ear operations calls for more care than it has been generally given, especially in young subjects; that measurement of blood loss is a feasible routine procedure in such operations; and that such routine measurements are of important clinical use.

ACETYLCHOLINE IN ANXIETY STATES.—M. S. Shaw has administered carbaminoylcholine chloride (doryl) to six patients suffering from acute attacks of anxiety. He reports a diminution in their subjective and objective symptoms during the administration of the drug, with a relapse on its cessation. He states that evidences of sympathetic overactivity elicited before administration of doryl were diminished during its exhibition. He concludes that his work offers further evidence in support of the hypothesis that the somatic symptoms found in anxiety states are referable mainly to imbalance of the autonomic nervous system.—*J. Ment. Sci.*, November, 1936, p. 785. Abs. in *Brit. M. J.*

INTERNAL SECRETIONS AND CANCER

BY PAULINE BEREGOFF-GILLOW, PH.G., N.S., M.D.

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WHILE at the New York Cancer Institute I had the opportunity of studying the blood of 300 cancer patients,[1] and I confirmed the work of Reding and Slosse.[2] I found that these patients have a low glucose tolerance and show a tendency to alkalosis and calcium deficiency. I believe, as did Reading, that these various changes are due to a perverted metabolism dependent upon the disturbance of the glands of internal secretion. My attention was then directed to probable changes in the blood of apparently normal persons with a history of an immediate family relationship to cancer. In 240 such cases with a history of cancer in at least 2 generations I found that 18 per cent manifested the same changes as did the cancer patients, suggesting a hereditary disturbance predisposing the individual to the influence of the secondary factor (irritation, trauma, mode of life, etc.), which might later cause active proliferation.

With the assistance of Dr. Yomans and others I attempted to treat a series[3] of inoperable cancer patients from the viewpoint of an endocrine unbalance. All the patients of this group had been given a life-expectancy of not more than 3 to 6 months. My method was to study completely the general condition of the patient; to eliminate foci of infection; to treat the anæmia; to increase the sugar and calcium metabolism, using glandular preparations according to the type of tumour and the area affected. Out of 15 patients treated, 5 were alive and well a little over 4 years; 4 died within 3 years; and 6 within two years. One case considered inoperable was operated on successfully three months after treatment was begun. The point I wish to stress is that we tend to neglect the general condition of cancer patients. I believe that our attention should be directed more toward the fact that cancer is a constitutional rather than local disease; that a possible cure lies in the proper and thorough scientific study of every case; and that we should not ignore the fact that the disease may be based on an endocrine disturbance. Recently many experiments have been conducted which prove that there is a definite relationship between the glands of internal secretion and the development of cancer.

Lassagne[4] increased tumours from 50 to 80 per cent in animals of 2 per cent cancer incidence, using repeated injections of œstrin. Zondek[5] definitely reduced the Ehrlich mouse carcinoma with prolan injections. Moller[6] confirmed the work of Zondek. The prolan-treated animals, at autopsy, showed a striking hyperplasia and hypertrophy of both adrenal glands, with microscopic changes corresponding to pregnancy. Connave[7] caused definite retardation of the growth of mouse carcinoma by injections of prolan before and after tumour inoculations. Ludwig and von Ries[8] confirmed his experiment. Hofbauer[9] produced a leukoplakia in the cervix of a guinea pig by repeated transplantations of fragments of the anterior lobe of the pituitary, while Overholser and Allen[10] produced epithelial hyperplasia in the uterine cervix of ovariectomized monkeys with injections of genital hormones. Geschickter, Hartman and Lewis[11] have shown a definite relationship of gynæcomastia, virginal hypertrophy, cystic disease of the breast, fibroadenoma and fibrosarcoma of the breast to the ovarian hormone œstrin. These investigators suggest a new method of diagnosis and treatment of these conditions.

It appears that malignant proliferation is produced more readily under an increased concentration of œstrin, while a high concentration of prolan tends to cause regression of the growth. As suggested by Zondek, it may be that the loss of prolan in the urine aids increased production of œstrin with resultant malignant changes. On the other hand, Halpern and D'Armour[12] have shown that the

pituitary gland is markedly hypertrophied after injections of œstrin, which may be responsible for the increased excretion of prolan in the urine of cancer patients. This antagonistic relationship between these hormones in the development of cancer is of great interest.

In 1929 I obtained from Dr. Segiura, of the Memorial Hospital, New York, a male mouse with spontaneous carcinoma of the breast. This I mated with females of a low malignancy stock, and the progeny were mated, brothers with sisters, back crossing of the young with their fathers and mothers. Within 4 years a strain developed which had adenocarcinoma of the breast in 50 per cent of the breeding animals (females) which lived till the cancer age. The tumours appeared between the ages of 5 to 18 months. Fifty animals of this strain were given approximately from 100 to 120 rat units of œstrin weekly. The number of mice in which tumours developed increased with the increase of œstrin injected. From the 50 per cent strain we developed a 85 per cent one.

Another group of 90 mice from our original strain was inoculated with mouse carcinoma. Forty were kept as controls, while 50 received 1,000 rat units of prolan before inoculation and 1,500 after. There was a definite retardation and inhibition of the tumour "takes" in those which received prolan as compared with the controls.

A number of recent investigators have obtained similar results. Most interesting are the experiments of Loeb[13] and Cori,[14] who by means of ovariectomy during early life in mice, prevented entirely the appearance of tumours in the majority of high tumour-rate strains.

Some time ago Hoffman, Parker and Walker[15] increased the growth of chicken tumour No. 1 with repeated injections of a rabbit testicular extract. Later, Duran-Reynals and Claude[16] confirmed their results. Matsuoka[17] showed that the growth of Kato rabbit sarcoma was increased by splenectomy and that various extracts of spleen inhibited the growth of this sarcoma. At the same time Inutsuka[18] inhibited the growth of sarcoma and carcinoma of rabbits by the injection of an extract of thymus. A partial extirpation of the parathyroid gland, he showed, increased the rate of growth of the tumours. Gwyer,[19] using large doses of thymus gland, diminished the pain and the size of the tumours, and greatly improved the general condition of the patient. Hanson,[20] using the same product, also obtained favourable results.

While Murphy and Sturm[21] decreased the rate of post-operative local recurrence after the surgical removal of spontaneous cancer in mice by the injection of extracts of desiccated homologous embryo skin and placenta, Lambert and Driessens[22] observed complete disappearance of Jensen rat sarcoma in 30 of 40 cases injected with insulin after the tumours had been grafted. Konsulov and Dimitrakow[23] inhibited the growth of spontaneous tumours and of Ehrlich mouse tumours by injections of insulin at the time of grafting. Boris Sakaloff[24] has demonstrated the liquefaction of tumours in mice and rats after injections of adrenal cortex, while Auler and Rubenow[25] produced regression of the Jensen sarcoma and Flexner-Jobling carcinoma in certain animals with injections of adrenal cortex.

It is true that several investigators working along similar lines have obtained negative results, but it is rather difficult in this connection to judge such results. One must not forget the great variation of tumours in animals and in human beings, and, most of all, the difference in potency of extracts should be considered before negative reports are relied upon. It is very easy to destroy the activity of hormones, and therefore every technical detail in their preparation should be most carefully observed. It is also to be understood that an extract may be effective in one patient and not in another with the same type of carcinoma. If there is no physiological relation between the gland and the area of growth an extract of such a gland should not be expected to have any effect on the tumour. For example, adrenal cortex extracts should have their best effect on carcinoma and sarcoma of the reproductive organs, except the breast, rather than on those of the tongue and face. Until the hormones are better known and their effects on growth noted by a large number of investigators differences of opinion will arise as to their value in clinical and experimental conditions. This, however, should not justify us in abandoning endocrine therapy in malignant diseases, especially when used in conjunction with present treatments. Let the research worker in this direction receive the same opportunities as the radiologist and the surgeon, for neither our marvelously equipped radium-x-ray departments nor sur-

gery have so far controlled this dreadful disease.

I am indebted to Dr. E. A. Sharp, of the Parke Davis Company, for supplying me with theelin and Antuitrin S, and to Dr. Harrower, Director of the Harrower Laboratory of Glendale, for a number of glandular preparations and for his interest in this work.

Note.—An extensive bibliography consulted for this paper is included in reprints and may be had on application to the author.

MODIFICATIONS OF MARSHALL'S CLINICAL METHOD FOR THE DETERMINATION OF URINARY UREA*

BY ANDREW HUNTER

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IN Marshall's "rapid clinical method" for determining urinary urea[1] the urine is treated with urease, and the ammonium carbonate produced is measured by titration of the mixture with acid in the presence of an appropriate indicator. From the gross result thus obtained the amounts of acid required to bring (*a*) the enzyme solution and (*b*) the urine itself to the selected end-point have to be deducted. Each of these corrections is determined by a separate titration.

The chief difficulty in this procedure is occasioned by the fact that at the rather acid end-point used the urine itself is clear, while the urease solution and the mixture containing both are turbid. The precise matching of the indicator in all three becomes under such conditions almost impossible. This difficulty would be avoided, and, at the same time, the number of titrations required would be reduced from three to two, if the total correction could be determined by titrating urine and urease not separately but as a mixture. To make this feasible it would be necessary that in the mixture serving as a blank the enzyme should in some way be rendered inactive. In the procedure lately described by Cole[2] this object is attained by utilizing the toxic action of mercuric chloride. A simpler and equally effective way of inactivating urease is to expose it for a short time to a moderate concentration of acid. According to Marshall himself[3] the activity of a soy-bean extract is permanently abolished by two minutes of exposure to 0.005 N HCl, and in experiments of my own a few seconds at a pH of 2.8 to 3.0 have sufficed to produce upon a jack-bean extract the same effect. Destruction of the enzyme by treatment with acid has this evident advantage, that it may serve as the first stage of, and accordingly be combined in one operation with, the blank titration.

As it was first described, the "rapid" method of Marshall had a second disadvantage—that it was inconveniently slow. This drawback was removed when Mateer and Marshall[4] discovered in the jack-bean a source of urease greatly superior to the soy-bean originally employed. As these authors showed, 2 c.c. of an active jack-bean extract require only half an hour at room temperature to decompose as much as 2.5 per cent of urea in 5 c.c. of urine. If only 1 c.c. of extract be employed the turbidity developing during the titration is reduced, and the sharpness of the end-point correspondingly increased; but, to secure the same performance, the time allowance must be extended to one hour. These conditions have been found to be adequate for the analysis of any ordinary urine. Thus, in one experiment, three 5 c.c. samples of the same rather concentrated urine, each treated with 1 c.c. of jack-bean extract, were titrated at the end of 1, 2 and 4 hours respectively. The volumes of N/5 sulphuric acid required were 23.48, 23.40 and 23.48 c.c. The blank was 2.11 c.c., so that the percentages of urea indicated were 2.56, 2.55 and 2.56. One hour, therefore, had sufficed to give a maximal result.

If both the main determination and the control are modified along the lines suggested one obtains a combination eminently suited to the routine analysis of a series of urines. This combination is described below as Procedure I. Although not heretofore described in print it has been in use in the writer's laboratory for many years, and has proved to be as reliable as it is convenient.

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In case it is desired to execute a single determination in the shortest possible time, one may increase the amount of enzyme extract to 2 c.c. (or even more), and, retaining for the blank the method of inactivation by acid, may adopt for the active mixture the technique of continuous titration which forms the second novel feature of the method of Cole.[2] This combination is described as Procedure II.

REAGENTS REQUIRED

Urease solution.—This may be a 10 per cent solution of commercial urease, or an extract of jack-bean flour such as that described by Schmidt.[5] To test its activity, add 1 c.c. to 5 c.c. of a 5 per cent urea solution, allow to stand at room temperature for 30 minutes, add a drop or two of methyl-red indicator solution, and titrate with N/5 sulphuric acid to the first shade of red. The amount of acid used should be not less than 15 c.c.

Sulphuric or hydrochloric acid, N/5.

Methyl-orange indicator solution (0.4 per cent in water).

Phenolphthalein indicator solution (1 per cent in alcohol). This is required only for Procedure II.

PROCEDURE I FOR DETERMINATIONS IN A SERIES OF URINES

Select two large test-tubes of as nearly as possible the same diameter (about 1 inch), label them A and B, and into each measure 1 c.c. of urease solution and 1 drop of methyl-orange indicator solution. To A add 5 c.c. of the urine to be analyzed; to B (which is to serve as the control) add about 10 c.c. of water. Close A tightly with a rubber stopper, mix its contents, and set both tubes aside for at least one hour. At the end of this interval proceed first with the determination of the blank. From a burette run into tube B, to begin with, just as much N/5 acid (usually less than 1 c.c.) as may be necessary to change the indicator to its deepest shade of red (pH about 3). After waiting for one minute, to ensure the complete destruction of the enzyme, add 5 c.c. of the urine. This will change the indicator back to yellow or light pink. Continue then the titration with the acid until the colour of the mixture is definitely, but not too deeply, red. Note the total amount of acid that has now been added to B.

Remove next the stopper from tube A, and at once titrate its contents with N/5 acid. Add the acid at first rather rapidly, so as to minimize any loss of ammonia by volatilization. When the indicator begins to change, proceed more cautiously. Add water, as required, either to A or to B, so that at the end the volume in each shall be as nearly as possible the same. The titration is finished when A makes an exact colour match with B. Under the conditions described the correct end-point can be estimated within one drop of N/5 acid.

From the result of the second titration (A) deduct that of the first (B). The remainder represents the urea decomposed; and, since the acid was N/5, each c.c. corresponds to 0.0028 g. of nitrogen or 0.006 g. of urea. Multiplication by the first of these factors will give the amount of urea nitrogen, multiplication by the second, the amount of urea in the sample taken. Since the sample measured 5 c.c., the percentage content of urea (or urea nitrogen) may be obtained by the use of factors twenty times greater, namely, 0.12 for urea and 0.056 for urea nitrogen.

PROCEDURE II FOR SINGLE DETERMINATIONS

Select, as in Procedure I, two identical large test tubes, A and B. Into each measure 2 c.c. of urease. Add to B (the control) 5 to 10 c.c. of water and a drop of methyl-orange solution; to A, 5 c.c. of urine and a drop of phenolphthalein solution. Owing to the rapid production of ammonium carbonate the mixture in A will presently begin to turn pink. As soon as this is evident, add from a burette, a few drops at a time, just enough N/5 acid to discharge the pink colour. Continue from time to time the addition of acid in such a way and at such a rate that the development of a definite pink is barely inhibited. By this procedure the escape of ammonia is prevented, and the mixture is kept at a pH (about 8) nearly optimal for the action of urease. After 5 to 15 minutes, depending on the original concentration of urea, it will be found that the rate at which acid must be added is diminishing. The reaction may then be hastened by warming the test-tube in a water-bath at 40 to 50° C., or even by simply clasping it in the hand. When at length, after 5 to 10 minutes more, no further tendency to become alkaline is perceptible, interrupt the titration, but leave the tube in the bath for 5 minutes longer. This will ensure the decomposition of the last traces of urea.

The interval of waiting is utilized for the determination of the blank. This is carried out with tube B in exactly the same way as in Pro-

cedure I, but, since the titration of A has not yet been completed, the additions of acid to B must be made from a second burette. (If this is considered inconvenient, the blank titration may be performed before the main one is begun. This entails, of course, a certain sacrifice of speed).

Having now allowed sufficient time for the complete hydrolysis of the urea, remove tube A from the water-bath and cool it under the water-tap. Add a drop of methyl-orange solution, and resume the interrupted titration. Continue the addition of acid until the colour of A exactly matches that of B. As the end-point is approached add water as may be required, either to A or B, until both are filled to the same level.

The result is calculated in the same way as in Procedure I.

RESULTS

Each of the procedures described above has been tested by comparison with the very accurate urease method of Van Slyke and Cullen.[6] Typical examples of the results obtained are shown in the accompanying table. It will be seen that in no case reported was the error of either of the clinical methods greater than ± 1 per cent. In another series the maximum error, encountered only once, was 1.6 per cent.

No. of urine	*Vol. of urine c.c.*	*Procedure followed*	——N/5 H_2SO_4 used—— *Main expt. c.c.*	*Blank c.c.*	*Difference c.c.*	*Urea found percentage*	*Urea according to Van Slyke and Cullen percentage*	*Percentage error of clinical method*
1	5	I	14.71	1.59	13.12	1.57	1.58	−0.6
2	5	I	14.14	1.46	12.68	1.52	1.51	+0.6
3	5	I	16.20	1.48	14.72	1.76	1.77	−0.5
4	5	II	20.10	1.78	18.32	2.20	2.19	+0.5
5	5	II	15.62	1.08	14.54	1.75	1.75	0.0
6	5	II	19.89	2.09	17.80	2.14	2.12	+1.0

I desire to acknowledge the assistance of Dr. George S. Eadie in the development and testing of Procedure I.

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Case Reports

A CASE OF STREPTOCOCCAL MENINGITIS TREATED WITH PRONTOSIL

BY GORDON C. DRAESEKE, M.D., F.A.C.S. AND EVERETT F. RAYNOR, M.D., C.M.

Vancouver

G.J., a girl of 14 years, was admitted to the Vancouver General Hospital on December 9, 1936. She had been treated for seven days previously for a discharging left ear. This had begun without pain in the ear. Three days before admission the discharge ceased and she developed pain in the mastoid area, with swelling over the tip of the mastoid, and she felt very miserable on that day. She had had previous trouble with the ear two years before, having had pain for two weeks but with no discharge. In September, 1936, she again had left earache, which cleared up with "drops" in about a week. A left mastoidectomy was performed on December 10th, and pus was encountered in the initial incision in the tip of the mastoid. The cells were well broken down in the tip of the mastoid. The lateral sinus extended well forward, and there was a space of only about half an inch between the upper knee of the lateral sinus and the posterior canal wall. The upper knee of the lateral sinus was exposed, the sinus being normal. The cells in the posterior angle and in the zygomatic area were not much affected; the dural plate was firm, and no dura was exposed.

The patient ran a normal course after operation, except for occasional headache, until December 16th, when she became nauseated but did not vomit; temperature 101.4° F. On sitting up she complained of dizziness, but there was no nystagmus, etc. The following day her condition was much the same; temperature normal. On December 18th the headache became more pronounced; temperature rose to 103.2° F.; there was very slight stiffness of the neck, and Kernig's test was only slightly suggestive. Lumbar puncture was performed; pressure, 16 mm. of Hg.; 8 c.c. of fluid were removed; cell count, 2,500, mostly polymorphonuclears. Short-chain streptococci were found in direct smear. On culture a hæmolytic type of streptococcus was found. Following lumbar puncture she complained of stiffness of the neck and felt very drowsy. Later in the day she became nauseated and vomited. Prontosil, 10 c.c. was given intramuscularly at 11 p.m. that night and again at 3 a.m., December 19th. Mastoid healed and dressings discontinued.

December 19th.—No further prontosil was given until 11 a.m. since we were short of supply, but after

that she received 5 c.c. intramuscularly every four hours, and she was also started on prontylin, gr. x, t.i.d., by mouth. The patient complained of severe frontal headache and felt very drowsy. Rigidity of the neck was marked and Kernig's test positive. A No. 6 ureteral catheter was inserted into the spinal canal for continuous drainage. Initial pressure of the spinal fluid, 22; cell count, 780. The catheter only drained spasmodically and the patient continued to complain of frontal headache. She was started on 500 c.c. of 5 per cent glucose in saline intravenously t.i.d., since the fluid intake was inadequate and she was vomiting frequently. The temperature varied between 102 and 103.2° F.

December 20th.—General condition much the same as on the previous day. The catheter was still not draining well. The patient was still nauseated and vomiting at intervals. Spinal fluid from the catheter showed a cell count of 780. She still complained of severe headache, especially on the right side of the head. Her temperature came down to normal and remained at that level for 10 hours, then rose again in the evening to 101.4° F. Prontosil, 5 c.c. was given intramuscularly, q.4h.; prontylin, gr. x, t.i.d.; 500 c.c. 5 per cent glucose in saline intravenously, t.i.d.

December 21st.—Condition much the same. Headache was still very severe; she was still nauseated, quite drowsy and listless. Since the catheter in the spinal canal was not draining well it was removed and lumbar puncture done. Pressure, 20; 6 c.c. removed; cell count, 810. The patient felt much more comfortable after spinal puncture and was able to take a little soup. The temperature fell slowly to 99 and rose later to 101° F. Prontosil, prontylin and intravenous injections as on the previous day.

December 22nd.—The patient was very irritable and objected to being moved. Stiffness of the neck was pronounced. Kernig's test was very definite. Lumbar puncture was attempted, but no fluid could be obtained. Cisternal puncture was resorted to; pressure, 22; 12 c.c. removed; cell count, 820. The patient took fluids a little better; no nausea or vomiting. Her temperature remained around 100° F. Prontosil, prontylin and intravenous injections as before.

December 23rd.—General condition much the same. She was taking fluids so well that we discontinued intravenous treatment. Since the spinal fluid showed no coloration we decided to introduce prontosil directly into the spinal fluid and 1 c.c. of prontosil was injected intracisternally. Cell count, 590. The patient showed no toxic symptoms from this injection; in fact she felt better and took more nourishment. Her temperature rose to 100.2° F. and then gradually fell to normal and remained so for 18 hours. Prontosil and prontylin as previously.

December 24th.—The temperature was normal until we injected 2 c.c. of prontosil intracisternally, when it rose to 101° F.; the patient felt a burning sensation in the head, became nauseated and vomited, and felt very dizzy. Her headache became very severe and she was very drowsy and listless. Her temperature remained elevated all day. Cell count, 325; pressure, 15; 8 c.c. removed; spinal fluid colourless. Prontosil and prontylin as before.

December 25th.—The patient was very much improved; headache less troublesome; no nausea or vomiting. She was able to move her neck. Kernig's sign, negative. She felt so well that she sat up and ate a small Christmas dinner of turkey. In view of the toxic effect of 2 c.c. of prontosil we decided to keep our intracisternal dosage at 1 c.c. Pressure 10; cell count, 295; the fluid was pinkish. The temperature varied between 98.2 and 100° F. Prontosil 5 c.c., intramuscularly. q.4h.; prontylin, gr. x by mouth t.i.d., as on previous days.

December 26th.—Still improving; she was able to brush her teeth and sit up for meals. One c.c. of prontosil was injected intracisternally. Pressure 10, cell count, 85; only a trace of globulin. Her headache was less troublesome; no stiffness of the neck; she was able to move her legs. Temperature, 97.2 to 99.2° F. Prontosil and prontylin as before. Spinal fluid colourless.

December 27th.—Because of marked improvement we decided to omit cisternal puncture and merely continued with 5 c.c. of prontosil intramuscularly every four hours, and prontylin, gr. x, t.i.d. by mouth as in previous days. Temperature remained practically normal all day.

December 28th.—Still showing improvement; cisternal puncture withheld. Treatment as before. Temperature about normal. Appetite was much improved. The patient complained of a dull headache on the right side of her head.

December 29th.—Patient moved freely in bed; no sign of meningeal irritation. Diagnostic cisternal puncture showed pressure, 12; cell count, 30. Twelve c.c. of fluid removed showed no growth on culture after 40 hours. The prontosil was cut down to 5 c.c. intramuscularly, t.i.d.; prontylin as before. Temperature between 97 and 99° F.

December 30th.—No complaints. We were able to move the patient's knees up to her head without discomfort to her. Treatment as on previous day. Temperature 98° F. all day.

December 31st.—The prontosil was cut down to 5 c.c. intramuscularly, b.i.d.; the prontylin was cut down to gr. v, t.i.d. by mouth. General improvement. The patient was able to read a book. No complaints. Temperature normal.

January 1st.—All treatment was discontinued. The patient strong enough to sit up in chair for 20 minutes. Since this time she has shown general improvement, with rapid gain in strength. No apparent after effects of the meningitis or treatment have been noted.

Laboratory work.—December 19th, white blood cells, 6,200; red blood cells, 4,500,000; Hgb., 75 per cent; December 22nd, white blood cells, 11,200; December 24th, white blood cells, 9,100; December 29th, white blood cells, 11,050; January 3rd, white blood cells, 11,200; red blood cells, 3,710,000; Hgb., 60 per cent.

CONCLUSIONS

1. A case of hæmolytic streptococcal meningitis with recovery, treated with prontosil, is reported.

2. Although there may be some tendency towards spontaneous recovery in some of these cases the dramatic response this patient exhibited, even after 24 hours' treatment with prontosil, cannot be overlooked.

3. Prontosil injected into the cisternal space gave no toxic results so long as the amount injected was small. A larger dose in our case produced a burning sensation in the head, in tense headache, nausea and vomiting, and an elevation of temperature.

4. Prontosil had no appreciable effect on the blood count in this case.

5. We tapered off the dose of prontosil because we did not know what effect abrupt cessation of the drug would have, but we feel it could have been discontinued earlier without gradual reduction in dosage.

AN ACUTE REACTION TO MERCUROCHROME IN AN INFANT

BY R. JULIAN BROWN, B.Sc., M.D.

Jasper, Alta.

Baby W.W., aged 5 months, was admitted to hospital on November 12, 1936, suffering with extensive first-degree burns. The infant had been having a medicated steam inhalation, when he upset the kettle of boiling solution. The burns covered the entire back, from the superior part of the occiput down to the buttocks, both shoulders, small areas on each arm, the neck from the line of the jaw to the horizontal nipple line, the left cheek, the right ear and auricular area. A small ring about the neck, deep in the skin folds, escaped injury. Butsein picrate dressings were applied until the child reached hospital, when tannic acid treatment was started.

The infant showed severe shock, but when this was under control made excellent progress, until a low grade infection developed under the coagulum about November 19th. This was treated by quartz light daily, and after twelve treatments had entirely cleared up. The eschar had peeled off naturally, and all the burned areas had healed except a small strip about the neck, on the chest and on the inferior portion of the occiput. These open areas were treated with a mild antiseptic ointment, but were healing very slowly.

On December 9th, mercurochrome ointment was started, in an endeavour to promote more rapid healing. The morning of December 10th showed considerable improvement in the open areas, but in the afternoon the mother, who, incidentally, is a nurse, noticed that the infant was very restless and extremely irritable. As the child was teething this did not attract undue attention. During the night he was very restless and began to show some fever, and at 4 a.m. of December 11th the mother noticed that the eyelids of both eyes were markedly swollen, particularly the left, for the eye could not be opened at all. At the same time a fine macular rash was evident all over the infant's body, together with several larger hyperæmic areas, some of which were as large as a dime. The temperature had risen sharply to 103.8° F., and the pulse and respiration to 140 and 44, respectively.

As the only change in treatment had been the use of the mercurochrome ointment this was discontinued at once, and the treated areas were hastily wiped clean. The child had been slightly constipated, so a normal saline enema was ordered, together with a small dose of saline laxative. The enema was returned with considerable formed stool, and the stool and liquid were a definite pink colour. Tests for blood were negative. Orders were left to obtain, if possible, a sample of urine, but unfortunately this could not be done, but the next two soiled diapers showed the same distinctive pink colouring as the stool and enema. Again the test for blood was negative. An enema later in the day, and all subsequent diapers were normal and did not show a trace of the pink colour.

The infant's condition rapidly and steadily improved, until by the evening of December 11th, his temperature was 99.2° F., pulse 120, and respirations 38. The swelling of the right eyelids had completely disappeared, but a slight degree of swelling of the left eyelids remained. The rash had entirely faded out and only two of the larger hyperæmic areas remained on the thighs. On December 13th all evidence of the acute reaction had disappeared and the child progressed rapidly to full recovery. The mercurochrome ointment, during its limited use, had evidently helped, for the healing of the denuded areas was much more rapid following its use than previously.

This was undoubtedly an acute reaction to the mercurochrome ointment, as all other factors in the treatment were the same as had been in use for some time previously. The fact that the stool and urine showed a distinctive pink colour indicated that there had been a very rapid absorption from the denuded areas, and also an attempt by the body at an equally rapid excretion. The rapidity of the excretion is further borne out by the very rapid recovery made by the infant as soon as the offending substance had been removed from the denuded areas.

GRANULOSA-CELL TUMOUR OF THE OVARY WITH PRECOCIOUS MENSTRUATION

BY R. B. HARE, M.B.(TOR.), F.R.C.S.(EDIN.)

Simcoe, Ont.

The small subject of the above condition with the long title, L.B., a girl of seven and one-half years, was first seen with the complaint of vaginal bleeding on July 17, 1936, the flow having started the previous day. There were no clots and the flow suggested menstruation to the mother. The child was normal in size and development, both physical and mental, for her age. She had no precocious sexual ideas apparently, but her breasts had enlarged somewhat and she had some pubic hair.

She was somewhat pale, but had been anæmic on several occasions when rectal bleeding had been occurring (due to rectal polyps). The first polyp was removed in November, 1931, when she was 32 months old. When she was four and one-half years old a small polyp was found, but it was not until she was five and one-half and rather anæmic again that, on sigmoidoscopic examination, two polypi were found in the lower rectum and one in the anal canal. Their removal was permitted and her recovery from the operation and the anæmia was uneventful.

The child was not seen again for bleeding till July of this year, when the vaginal bleeding began. Rectal examination (digital) at this time showed another polyp about 3 inches up the rectum, and some enlargement of the uterus, but examination was not satisfactory.

She was given iron for her anæmia, and the vaginal bleeding stopped in 12 days. Examination under anæsthetic was then carried out and the right ovary was found to be quite enlarged and the left scarcely palpable. The uterus was enlarged to about twice the normal size for her age. The vagina was inspected by means of an urethroscope, in case a polyp were present, and no abnormality was found in the vagina or cervix. The polyp in the rectum was removed by diathermy after sigmoidoscopic examination had revealed no other polypi. Iron therapy was continued and laparotomy advised, but was not accepted by the parents till after she had menstruated again for two weeks, August 5th to 19th.

Laparotomy was carried out on August 26, 1936. The right ovary was found large and smooth and yellowish pink, and of the size and consistency of an adult testicle. The other ovary seemed normal. In view of the number of polypi found in the lower bowel the opportunity was taken to palpate the rest of the bowel, but no masses were found in the colon, but on picking up the small bowel one loop felt very boggy and thickened, and on delivering it from the wound an intussusception was discovered, the mass being about 6 inches long. This must have been very recent as there was no congestion or infiltration and no vomiting or pain before the anæsthetic was begun, and the intussusception was reduced quite readily. At the head of it was a thickening and dimpling which proved to be a polyp in the lumen of the bowel, the mass being about three-quarters

of an inch in diameter. No other masses were found.

The bowel was opened along the antimesenteric border by an elliptical incision, removing the tumour and its base, and was sutured transversely. The right ovary and tube were removed and the wound was closed without drainage. Convalescence was quite comfortable, but the girl flowed quite profusely for a few days and then the flow gradually decreased and ceased in about two weeks.

Pathological report on right ovary and tube.—"Gross specimen consists of a fallopian tube and ovary. The fallopian tube is moderately tortuous. It is pinkish-grey in colour, measuring 6.8 cm. in length by 8 mm. in diameter. The fimbriæ are free. A small accessory ostium was found mid-way along the fallopian tube. The ovary was somewhat spherical in shape, measuring 5.5 by 4.8 by 4 cm. The ovary was almost entirely occupied by a large solid opaque golden yellow tumour nodule, measuring 3.9 cm. in diameter. This tumour simulated a large solid corpus luteum of pregnancy, save that it was larger and no hæmorrhagic centre was present. One or two small cysts were found in the adjacent ovarian stroma, only a very narrow rim of which was found about this tumour nodule.

"*Microscopic report.*—This tumour was composed of many gland- and cord-like structures lined by epithelial cells. These cells tend to be rather large, possessing very pale staining vacuolated cytoplasm and relatively deeply staining nuclei. The glandular structures for the most part are quite well formed, and some of them contain small granules of calcareous material within the lumina. Sections stained to demonstrate fat show these epithelial cells to be heavily laden with large droplets of fatty material. The histological character of the cells showing them to be quite uniform in size and shape throughout does not suggest malignant qualities possessed by this tumour. The predominating type of cell is that found in the granulosa-cell tumour and in the sections studied there is no evidence of breaking through of the capsule into the adjacent tissue. The sections of fallopian tube present nothing remarkable."

Pathological report on the material from the small bowel.—"Grossly, this consists of a polypoid mass of pinkish-grey tissue, of velvety character, measuring 3.8 by 2.4 by 1.7 cm. The mucosal surface throughout is coarsely velvety in character showing no gross evidence of ulceration. The pedicle of this polyp is quite narrow and shows no infiltration or induration.

"Microscopically, this fragment is definitely polypoid in character, and the free surface is clothed by mucosa of glandular character such as is found in small intestine. In the deeper layers of the mucosa the epithelial cells are formed in a glandular pattern suggesting an adenomatous character. A moderate grade of subacute inflammatory infiltration is present throughout the stroma. The epithelial cells throughout the section are quite uniform in size and shape, presenting no invasive characters. There is no evidence of malignancy. Diagnosis.—Adenoma of the small intestine (polypoid)."

In going back into her history the child had had an attack of colicky abdominal pain with vomiting at twenty months, lasting six hours, and again at five years she had an attack of pain, vomiting and blood in the stools. This time the polyp in the rectum was blamed for the bleeding, but it may have been a temporary intussusception at that time.

The ovarian tumour is a rare type referred to as granulosa-cell tumour. To save repetition, the reader is referred to a rather extensive study of these tumours by Blaud and Goldstein in *Surgery, Gynæcology and Obstetrics*, 1935, **61**: 250. These authors summarize 160 cases of which only 8 occurred in children under 11½, and in 2 of these the growth recurred locally after removal.

This child also has quite a degree of melanin pigmentation of her lips which has been present from birth. One wonders what other abnormalities may show up as time goes on. She is the only girl in the family, and there are three brothers, all of whom seem quite normal.

SPONTANEOUS RUPTURE OF A STRANGULATED HERNIA

By W. F. Gillespie

University Hospital, Edmonton

On July 17, 1933, S.K., a 72-year old Polish farmer, was admitted to the University Hospital with a history of a right inguinal hernia, (first noticed eighteen months previously), suddenly becoming incarcerated, swollen and tender 11 days before admission. He was immediately in great pain, vomiting almost constantly, and having watery stools. On the 5th day the lump ruptured through the skin, pouring liquid fæces over the belly. Following this there was no pain, save for the burning about the fistula. But he felt himself growing progressively weaker, and was exhausted from the frequent watery stools passed per anum as well as the fistulous drainage.

Examination showed an emaciated, senile, dehydrated man. On the scaphoid belly was a large fistulous opening in the right groin, surrounded by an extremely excoriated skin over an area 7 inches in diameter. At the margins the skin and superficial fascia hung in foul, gangrenous shreds. Thin fæculent fluid bubbled out intermittently. General examination showed a large, irregular heart, pipe-stem arteries, a blood pressure of 80/50, and moderate renal insufficiency.

Two weeks were spent in improving the patient's general condition, and relieving the skin condition. On August 2nd a right paramedian incision was done under block anæsthesia. Slight matting of intestinal loops about the right internal ring was found. This was freed enough to permit a lateral anastomosis about three inches from the fistula. Following this the patient greatly improved both in weight and in strength. The bowels moved daily, but still there was copious irritating discharge from the fistula, which defied plugging. A second operation was done, at which the two loops of small bowel between the site of anastomosis and the fistula were completely divided, and the four stumps inverted. The patient did well after operation except for slight wound infection. He was discharged twenty-six days after this, feeling well, having a healthy abdominal wall, and a moderate amount of bland mucus from the excluded intestinal fistula. The last progress note (July 20, 1936) states that he feels quite well, except for the continuing watery discharge.

Editorial

ON TO OTTAWA

IT will soon be time for the medical literati and virtuosi to forgather for their annual bonspiel. This year the pilgrimage is to Ottawa—the Capital City. The Canadian Medical Association has not met in Ottawa since 1924. Those who were at that gathering well remember the excellence of the scientific provision and the hospitality of their hosts. The Meeting this year promises to surpass the former, perhaps all former meetings, if enthusiasm and industry can be taken as criteria. Our Association and the Ontario Medical Association will be the guests of the Ottawa Medico-Chirurgical Society—a happy augury. The palatial Chateau Laurier will be the headquarters and is ideally adapted to our purpose. A glance at the Scientific Program will convince one that it is of a high order. The British Medical Association, the great medical body with which we are affiliated, sends two of its members to take part. This is a gratifying gesture and these gentlemen will be welcome. The success of the Scientific Exhibit of the Ontario Medical Association last year set a precedent which the Ottawa Committee has not been slow to follow, with the laudable hope of surpassing it. The Commercial Exhibits promise to be the best in many years.

The Blackader Lecture will be delivered by Dr. H. B. Cushing, of Montreal.

His Excellency Lord Tweedsmuir will be the guest of honour at luncheon on June 23rd, and will also take part in the ceremonial gathering (which will be something to see) on the evening of the same day. The Premier, the Right Honourable W. L. Mackenzie King, will speak at one of the luncheons, also.

The Social Functions will be outstanding, notably a Garden Party at Rideau Hall. The Ladies will be well taken care of, as usual, and a particularly happy thought was the provision for the Juniors of the party, who will be specially entertained. It is felt that by bringing the children to the Capital City interest in the historic side of our country will be stimulated and high national and imperial ideals will be inculcated.

At Ottawa, in the week of June 21st, there will be something of interest for all, so

ON TO OTTAWA.

THE METABOLIC FACTOR IN ACUTE RHEUMATIC FEVER

IN the case of a disease so complex and badly understood as rheumatism it is not surprising that a great amount of labour has been expended in an attempt to elucidate its metabolism—an amount, it may be said, quite disproportionate to the meagre results that have been obtained. The chronic forms of arthritis seem to have claimed the most attention, but acute rheumatic fever is gradually coming into the limelight.

As far back as 1858, on the basis of experiment, Richardson[1] suggested that rheumatism was due to an accumulation of lactic acid in the body. In 1877 Foster[2] reported that the administration of lactic acid by the mouth to two diabetic patients resulted in painful and swollen joints. These manifestations persisted so long as the lactic acid was continued and disappeared when it was discontinued. These experiments were apparently not followed up, but, nevertheless, the lactic acid theory of the causation of rheumatism for years after had many supporters. Within recent years Wilde[3] has revived the theory, chiefly on clinical grounds. Pemberton[4], however, at

1. RICHARDSON, B. W.: The Cause of the Coagulation of the Blood, John Churchill, London, App. III, 1858, p. 371.
2. FOSTER, B.: Clinical Medicine, J. & A. Churchill, London, 1874, p. 143.
3. WILDE, P.: Physiology of Gout, Rheumatism and Arthritis, Wm. Wood & Co., New York, 1922.
4. PEMBERTON, R.: Arthritis and Rheumatoid Conditions, Lea & Febiger, Phila., 1935, p. 113.

least in the types of arthritis studied, in no case found abnormal quantities of lactic acid in the blood, urine and sweat.

Rinehart and Mettier[5] seem to have been the first to suggest that acute rheumatic fever is in some way linked up with deficiency of vitamins, and, with their collaborators,[6] have produced evidence to show that vitamin C is the one at fault. They think, however, that to the factor of deficiency of vitamin C infection must be superadded. Guinea pigs subjected to experimental scurvy and then to infection acquired cardiac, articular and other lesions markedly resembling those of rheumatic fever. It would seem from this that, inasmuch as rheumatic fever often attacks undernourished children, latent or "masked" scurvy may be a factor in predisposing to rheumatic infection. Some of the work done on vitamin C deficiency has been discounted on the ground that the "capillary resistance" test, otherwise called "the Rumpel-Leede phenomenon", is not a reliable test, being found in other conditions that scurvy, and that the chemical tests used in the earlier work for determining the presence of ascorbic acid in the secretions and excretions are also faulty. C. B. Perry, in some recent work,[7] based on a study of eleven cases of rheumatic heart disease, tested by administering ascorbic acid and then measuring the amount of ascorbic acid in the urine, while admitting that in some cases of rheumatism there might be a slight deficiency of ascorbic acid, came to the conclusion that this deficiency was not an important factor in rheumatic children.

Other negative evidence, this time in connection with vitamin A, is afforded by the observations of Stimson, Hedley and Rose,[8] who found that in animals rendered defincient in this vitamin superadded infection did not produce significant lesions. In this, as in other phases of experimental work connected with rheumatism the evidence produced is conflicting. Much more work will have to be done before a final conclusion can be reached. It is not impossible, of course, that deficiency in vitamins may in some cases be invoked, as are many other influences which make for the lowering of health standards, but this need not be invoked as the all in all in the etiology of rheumatic fever. Infection still seems to be the most important factor; all others are merely predisposing or contributory. A.G.N.

5. RINEHART, J. F. AND METTIER, S. R.: The joints in experimental scurvy and in scurvy with superimposed infection, with a consideration of the possible relation of scurvy to rheumatic fever, *Am. J. Pathol.*, 1933, **9**: 952; *ibid.*, 1934, **10**: 61.

6. RINEHART, J. F., CONNOR, C. L. AND METTIER, S. R.: Further observations on pathological similarities between experimental scurvy combined with infection and rheumatic fever, *J. Exper. Med.*, 1934, **49**: 97.

7. PERRY, C. B.: Rheumatic heart disease and vitamin C, *The Lancet*, 1935, **2**: 426.

8. STIMSON, A. M., HEDLEY, O. F. AND ROSE, E.: Notes on experimental rheumatic fever, *U.S. Pub. Health Rep.*, 1934, **49**: 361.

THE HEALTH OF THE COLLEGE STUDENT

IN previous issues of the *Journal* we have dealt editorially with the health of the college student and the methods by which college authorities are endeavouring to deal with that problem.[1] We make no apology for returning to the subject. It is important; the colleges are alive to their responsibility; the students stand to gain.

There is a growing feeling among educationists that a college education is not for everybody. It should be obvious that students who previously have been badly trained or who exhibit no intellectual promise cannot profit much from a college course. They are, in fact, cumberers of the ground. We should add that those whose state of body or mind is such that they are unlikely to be able to complete the curriculum, or if they do so do it at the expense of health and perhaps at the cost of life itself, are an economic liability to themselves, their relatives, and their college. College authorities, therefore, owe it to themselves, to their students, and to the community to look after the health of their wards to the best of their ability. How fully they meet their obligations depends not only on their sense of responsibility but also, unfortunately, on the depth of their purses. We, in Canada, cannot hope at the moment to duplicate the

1. Edit. *Canad. M. Ass. J.*, 1936, **35**: 313 and 553.

very elaborate and comprehensive programs inaugurated by some of the larger American colleges to safeguard the health of the students. Nevertheless, much good work has been done, the value of which should not be underestimated.

Since our previous references to this subject much information has come in from our Canadian institutions so that we can now have a more accurate appreciation of our own domestic situation. For this information we are greatly indebted to Drs. A. S. Lamb, Director of Physical Education, McGill University; George D. Porter, Director of Health Service, University of Toronto; W. Ford Connell, Medical Officer, Queen's University; F. J. H. Campbell, Dean of the Medical Faculty, University of Western Ontario, A. T. Mathers, Dean, University of Manitoba; W. S. Lindsay, Dean of the University of Saskatchewan; Allan C. Rankin, Dean, and Egerton L. Pope, Professor of Medicine, University of Alberta, to whom we extend our thanks.

The impression obtained on reading the statistics available for certain of the larger American universities is that tuberculosis is somewhat more common among college students than among the general population, and more common among medical students than among others. The figures for this disease, too, are higher for students haling from the more congested centres. It should be said, however, that as complete statistics covering the various points are not available, and perhaps never will be, the impression remains only an impression. In the case of our Canadian universities, so far as our information goes, tuberculosis appears to be far from common, we are glad to say. Taking the figures for the University of Toronto, which are the most complete of any before us, Dr. George D. Porter states[2] that 9,968 students were medically examined during the past eleven years. Among these was a history of pleurisy in 193 (2 per cent) and of tuberculosis in 36 (0.3 per cent). Among first and second year students (during a period of eight years) acute pleurisy was found 8 times and active tuberculosis only twice. Indeed, the record of the Toronto students in the matter of health is remarkably good. At the time of their examination only 2 per cent were found unfit to take physical training or engage in athletics in any form. Five per cent had only such disabilities that light corrective exercises were required, and the remaining 93 per cent were able to take part in various forms of athletics or gymnastics. An analysis of those unfit for physical training or sports showed that one-half of them were crippled because of heart trouble and two-fifths of this half gave a definite history of rheumatism. Enlargements of the thyroid were noted in 667 (approximately 6.5 per cent) and hyperthyroidism in 29 (about 0.3 per cent). At the University of Alberta only 8 cases of tuberculosis were detected among the whole student body during the past thirteen years. The incidence of rheumatism and heart disease there also appears to be very low. The Prairie Provinces would naturally be expected to have low rates for tuberculosis, if we except the Indians, and few of them go to college.

All the Canadian universities are giving attention now to the health of their students. In none, perhaps, is that attention as complete as could be wished, no doubt for financial reasons. The will to do is there, nevertheless, and good results are being obtained. It is quite impossible in the course of a short notice to go fully into the various plans that are being operated, but we shall refer to the salient features of some of them.

Generally speaking, all new students are examined medically on entrance to the university. In institutions where physical training is compulsory the students are examined on entrance and, usually, at the beginning of the second year. Such examinations are reasonably complete, but except in special cases are not carried over to the later years. X-ray and tuberculin tests are only conducted when they seem to be indicated. At the University of Alberta all medical students are given a Mantoux test during their fifth year and non-reactors are asked to have the test repeated during the final year. At the University of Saskatchewan, with some assistance of the Saskatchewan Anti-tuberculosis League, all students are tested with tuberculin, and the

2. PORTER, G. D. AND MACLEAN, D. L.: Physical findings in young adults, *Canad. Pub. Health J.*, 1932, **23**: 416.

reactors are given clinical and x-ray examinations. Small-pox vaccination is required in some colleges on admission.

As the object of physical examinations is to promote health and bodily fitness as well as to detect defects and disease the measures just referred to are supplemented in some institutions by lectures on hygiene and by practical classes. In the University of Toronto special classes for corrective work and for those unable to undertake strenuous exercise are provided. Students may take to work of the Officers' Training Corps in lieu of gymnastics should they so desire. All students are taught to swim, and many of them take the course on life-saving. In the majority of cases improvement of the physical standard of the Second Year student is found, as compared with that in his First Year. Lectures on First Aid are given to the First Year Medical students and to those in Forestry, and occasional lectures on hygiene are given to various groups. Three lectures on Personal Hygiene are given to First Year students.

At McGill, Saskatchewan and Alberta, for a yearly fee of $5.00 students are provided, under prescribed limitations, with medical advice and attendance, and a limited amount of hospitalization. This idea is, of course, amenable to great expansion.

Through all these measures the students are being educated to become "health-conscious", and there is no doubt that, more and more, thay are becoming appreciative of the value of good health and of the means necessary to preserve and promote it. Despite the incompleteness of the health programs in operation in Canadian colleges we can say that the results obtained have been well worth while. A.G.N.

Editorial Comments

British Health Resorts

"British Health Resorts", the official handbook of The British Health Resorts Association, of which our old friend, Dr. Alfred Cox, is the energetic General Secretary, is again before us. It is a useful visitor, as it gives in an attractive form much information in regard to spas and health resorts, not only in Great Britain but in Canada, Australia, New Zealand, South Africa, and other parts of the British Empire. In fact, it seems to be the only book of its kind. The various resorts are briefly characterized and their attractions emphasized by excellent pictures. The whole is introduced by scientific disquisitions on the place of climatology in the treatment of disease. Certain shortcomings in regard to the provision in Britain for invalids are being overcome, and it is safe to say that, even now, it is unnecessary to send our patients who are in need of rest, massage, baths, physical therapy, and a sojourn in the open amid delightful and health-giving surroundings to a foreign country. British Health Resorts are now the peers of any.

This volume contains some new information. There is a valuable chapter on climatology, containing some very suggestive reflections. There is a synopsis of the attractions offered by British Resorts during the "invalids' winter". Additional information is given as regards the rainfall and sunshine in many resorts. The latter is divided into the fall during the day and during the night—an important point from the visitors' point of view. Much information as to health and health resorts is to be found here.

The book is published by J. & A. Churchill, Ltd., 104 Gloucester Place, Portman Square, London, W.1., and the price is two shillings and sixpence net. A.G.N.

A New Hospital Magazine Launched in Australia

Medical men and others interested in hospitals in Victoria, Australia, will welcome a new hospital journal entitled *The Hospital Magazine*. This will be published by the Charities Board of Victoria and will be under the editorship of the Chairman of that Board, Dr. J. Newman Morris, whose name has long been associated with medical and hospital progress in Australia, and who is personally known to many Canadians. In its initial number *The Hospital Magazine* carries a wide selection of articles on divers hospital topics, several being written by well known medical authorities. To this new journal we extend our most sincere good wishes. HARVEY AGNEW

Corrigendum

Dr. R. H. Macdonald wishes us to state that an error occurs in his case report entitled "A Case of Pharyngo-œsophageal Diverticulum", which appeared in the May *Journal*. On page 518, first column, line 18, "5 per cent procaine hydrochloride" should read 0.5 per cent.

Special Articles

SOME CAUSES OF CANCER IN LABORATORY ANIMALS*

BY C. LEONARD HUSKINS

Department of Genetics, McGill University, Montreal

If there is one thing that ought to be obvious it is that heredity and environment are interacting, not opposed or independent, forces. Yet one is continually being asked which is the more important, and there is continuous opposition between some geneticists and some environmentalists. This is particularly evident in certain recent discussions of the cancer problem. I have therefore, since accepting your President's invitation to speak to you on the cancer problem, decided that I shall really talk about heredity and environment, using the cancer problem as an illustration. I shall set forth for you genetic and environmental views about the cause of cancer which show that both must in part be right, and that in this case at least it matters very little to you as practical medical men which is the more right. I shall say nothing about the development or treatment of cancer; about those aspects I know that I know little.

As long ago as 1891 von Hansemann and Boveri found that in cancerous tissue many of the cells have irregularities in chromosome number or behaviour. Boveri, in experiments on sea-urchin eggs, was the first to prove that the different chromosomes carry different hereditary factors, and that a full complement of chromosomes (and of hereditary factors) is necessary for normal development. Following this initial work we have had a long series of papers attempting to prove that chromosome aberrations are the cause of cancer. Unfortunately, that hypothesis is too simple to be tenable today. We now know that the gross chromosome aberrations are an effect rather than a cause.

We can likewise dismiss as too simple the view of Slye that a single recessive hereditary factor is the cause of cancer, though this remark must not be construed as derogatory or as minimizing the significance of her pioneer work; I simply mean that we have found the genetic aspects to be much more complex than she assumed.†

On the environmental side we can at once dismiss the earlier ideas that cancer is due to microscopic organisms — to animal parasites, bacteria, etc. On the genetic side the view which holds the field today is that cancer is due to somatic mutation, that is, to a sudden and potentially permanent change in the hereditary material of the body cell. We can, perhaps, best date this somatic mutation theory back to Oertel (now at McGill) and Ross, who in 1905 pointed out that malignant growths never arise in normal tissue without preparatory stages preceding them. There must be irritation (using that word in a general sense) for a long time before a change to malignancy occurs. The most complete evidence for the somatic cell mutation theory of cancer seems to me to come from the work of Curtis, Dunning and Bullock, of the Crocker Laboratory, New York. They have shown from their work on rats infested with *Cysticercus fasciolaris*, the larval form of the common tapeworm of the cat, that though this may produce in the host rat from one to one hundred benign cysts, malignancy is usually confined to a single cyst. They report further.

> "Of 4,321 cysticercus tumours 79.3 per cent were sarcomata of the large-cell type and probably arose from the cells which formed the inner zone of the cyst wall; 20.6 per cent were sarcomata composed of smaller cells and possibly arose from the outer zone of the cyst wall; 6 were adenomata; and 1 was a carcinosarcoma which must have arisen from the snared-off bile duct and liver epithelium which was embedded in the wall of the cyst." They conclude that "If the change of a normal to a tumour cell results from a process analogous to somatic mutation, which is brought about by the chance action of an irritant, it would be expected that the types of cysticercus tumours observed would represent the various types of cells found in the cyst wall and that each would be represented by a number proportional to the comparative number of these cells exposed to the irritant and their relative proximity to it. The above classification (see their Table IX) shows that they roughly approximated this expectation."

On the environmental side the field is largely held today by those like Gye, Andrewes, Rous, Shope and others, who hold that viruses are the cause of cancer. Shope, some four years ago, found that a benign papillomatous tumour of the cotton-tail rabbit could be transferred to the domestic rabbit, and that a filtrate from these tumours could also transmit it. It was found that the virus (for such we must assume it to be) can affect only epidermal cells and that it causes specific types of tumours. The virus will withstand desiccation or preservation in glycerol for considerable periods of time and still retain its virulence. The growth the virus produces is not malignant; in fact Andrewes, of the British Cancer Research Institute, says "Few if any pathologists would consider them to be true tumours" (evidently

* Invitation address to the Royal College of Physicians and Surgeons of Canada, Ottawa, October 31, 1936.

† Since this was written Dr. Slye has announced (A.A.A.S. Meeting, December, 1936) her acceptance of the more complex modern genetic viewpoint.

by tumours he means malignant growths). Independently, Shope and Andrewes found a change or mutation in the virus; the new changed type produces inflammatory lesions. Rous, in an article in the *American Journal of Cancer* uses his original fowl virus and this rabbit virus of Shope's to argue that viruses are the specific cause of cancer in general. He sets up a number of arguments he assumes might be raised against his point of view, and proceeds to demolish them. He does not, however, raise as severe arguments against his point of view as others might do. His most telling point, in my opinion, is the fact that the papillomata will apparently regularly form cancers if given long enough, and that two distinct forms of this rabbit virus have now been separated. Malignancy arises by gradual changes, which he says are gradual changes of the virus. He says that though it looks like somatic mutation it is *not*, because if it were we should have to postulate a series of mutations of a kind not hitherto known. All mutations, even lethal ones, he says, are still "organized". In saying this, he quotes the elder Haldane who was a physiologist and not a geneticist, and that statement today is definitely wrong. Mutations of this type have become known during the last nine years. In a number of plants genes are known which cause asynapsis, that is, failure of chromosome pairing at the time of germ cell formation, and the result is unorganized and irregular development of non-functional germ cells. In maize there is a gene which produces polymitosis, that is, after maturation divisions have occurred the cell keeps on dividing, until finally there is a mass of cells with only one or a few chromosomes each, instead of a normal complement. In maize also Jones has shown that small tumour-like growths on the seeds are often associated with mutations of other characteristics, such as colour on the seed-coat. This only shows how dangerous it is to base conclusions on a negative statement in a science expanding as rapidly as is genetics. But the most serious objection to Rous' views that I see is that he has not yet shown that the changes which he postulates in the virus cause that virus to be any more virulent or more capable of producing malignancy directly than is the original papillomata-producing virus. Of course, if he does show such a change this statement will be out of date.

Now let us turn to some genetic evidence of a different type which has been used to show that the origin of tumours is genetic. In the fruit fly Drosophila there exists a gene the presence of which causes all individuals possessing it to develop small black tumour-like growths. Whether they are malignant has not definitely been established; the gene which determines them has been located as occupying a definite position in one of the chromosomes—the one which carries also the genes predominantly determining sex. In man there is the condition Xeroderma pigmentosa produced by a single gene; it is of course not malignant. In the platy fish there is a gene which determines the occurrence of black pigmented areas on the skin. In hybrids between the platy fish and the sword-tail this gene, acting in conjunction with the sword-tail germ plasm, produces melanotic tumours in place of spots. If crosses are made between two species of the tobacco family, tumours resembling those produced by *Bacterium tumefaciens* but which are not due to a pathogenic organism arise spontaneously on the progeny. Sometimes they are so numerous that the whole top of a plant becomes tumourous and normal growth is prevented. This condition can be made hereditary in certain progeny of the hybrids through doubling of the chromosome complement. These cases, it seems to me, can be set up against the two cases of virus in the fowl and in the rabbit, which are stressed by Rous. But, to turn again to the environmental causation of cancer. Besides the rabbit and fowl viruses there is, as is well known, a series of hydrocarbons—of tar and tar derivatives—which will, after repeated application, produce malignant neoplasms. In one case it has recently been shown that the neoplasm so produced can be transmitted by its filtered extract. Collip and his co-workers have also showed that œstrin can cause metaplasia and benign tumour formation in the uterus of the rat. The cysticercus secretion can apparently affect any type of cell. Should we, on Rous' view, therefore, consider it a more potent type of virus or are Rous' viruses more limited carcinogenic agents than this or the hydrocarbons?

The virus-produced papillomata are not malignant, though when they either naturally or artificially become deep-seated they may produce malignant growths. Rous concludes that the virus has *caused* the malignant neoplasms, though until recently he more cautiously stated that "the virus is the *primary* cause—the *proximal* cause is still to be determined." Rous notes that the malignant tumours so produced have varying characteristics, though, being derived initially from epithelium, the range of variation is limited. Some of us wonder whether the virus is not to be considered merely as another inciting agent and not as a specific cause in the sense in which Rous thinks it. About viruses as a whole we wonder greatly since the recent discovery that tobacco mosaic virus can pass through crystalline chemical form and still do its specific job thereafter. Though viruses reproduce themselves, they appear to do so only when asso-

ciated with organisms which are "alive" in the ordinary sense of the term. But suppose we assume that Rous is proved to be right and that viruses are the cause of cancer in the same definite (yet none the less limited) sense that the tubercle bacillus is the cause of tuberculosis. Will even this greatly lessen the significance of genetic constitution in the causation of cancer? Will it show environment more important than heredity? Shope has shown for instance that if a certain mixed virus, composed of the original and the new strain, is inoculated into the testis of rabbits both testes of any one individual will show the same reaction, but some rabbits will have fibrous tumours and others will have acute inflammatory lesions, and from these, in turn, the specific virus for its type can be isolated. The genetic constitution of the rabbit thus determines the kind of tumour it develops. It is questionable if papillomata can be continued by transplantation—it seems rather to be the transplantation of virus, but the eventual cancers can be transplanted. The upholders of the virus view as a general explanation have to postulate the *universal* occurrence of the virus so that from their own evidence it looks as if genetic constitution might be the more *important* thing. In discussing heredity and environment in general I usually state that it is meaningless to say which is more important unless one states the conditions of the experiment or observation, and then, after showing how the action of the two can not be separated, go on to say that what man must do is to change the component which is the most readily changeable. In plants we breed for disease resistance; in animals and man we usually try to eliminate the environmental factor, the organism causing disease. If the virus people are right, and the virus is everywhere, we are in cancer driven almost to an impasse, since it is scarcely practicable either to breed for resistance in man nor is it possible to eliminate the organism. I hope that Rous is not right. It is easy to give up smoking if one belongs to a family susceptible to cancer of the mouth, and most of us can avoid tar. Women of breast-tumour families can have frequent examinations, and, as in mice, we may be able to lessen the chance of cancer by hormone treatments and operations—but how is one to avoid a universally present virus?

But let us turn now to a consideration of some specific genetic data. Dr. Strong at Yale, for instance, has an inbred line of mice known as the C3H strain: in the last 42 generations of brother and sister matings 38 of the females in direct descent have died of mammary carcinoma. In the last 21 generations, which comprise the period when one may feel fairly sure that the strain has become purified for all genetic factors, every one of the 21 females in direct line of descent has died of mammary carcinoma. And in 15 years none other than mammary cancers have been found in this strain of mice. On the other hand, Dr. Strong has strains of mice in which cancer occurs with extreme rarity, though it is doubtful if any can be considered absolutely resistant. He has, however, nine or ten strains showing widely different degrees of susceptibility or resistance.

C. C. Little, at Bar Harbour, has Strong's strains of mice and others which are similar. One of his most significant contributions has been the demonstration that females transmit mammary carcinoma to their offspring to a greater extent than males do. In a cross between a susceptible mother and a resistant father 40 per cent of the female offspring have mammary carcinoma. In the reciprocal cross only 6 per cent have it. Evidently there is something transmitted in the cytoplasm of the egg as well as genes on the chromosomes which effects cancer susceptibility. I should add, however, that one has to bear in mind that in Drosophila similar cases of "maternal inheritance" have been proved to be due to chromosome genes affecting the cytoplasm in one generation and the cytoplasm passing this effect on in the next.* However that may be, this complexity discovered by Little is one of the things which lie behind my earlier statement that Maud Slye's view is too simple.

One of the most striking discoveries made on the cancer problem in recent years is that of McDowell, Taylor, and Potter. They have strains of mice highly susceptible and highly resistant to inoculation by certain strains of leukæmic cells. It was found that embryonic tissue macerated and injected into *one* susceptible strain would cause complete immunity to inoculation by *one* line of leukæmic cells.

Strain	*Treatment*	*No. of mice*	*Result*
Susceptible	Inoculated with leukæmic cells without prior treatment	104	All die
Susceptible	Stoli embryo suspension injected, then inoculated with leukæmic cells ..	93	All live
Susceptible	Susceptible embryo suspension injected, then inoculated with leukæmic cells	75	All die
Susceptible	F_1 Embryo suspension, then inoculated	80	All live

Unfortunately this 100 per cent resistance to leukæmia depends on a specific reaction; the Stoli embryo will *not* immunize any but the *one* strain. With human beings as

* Little and his co-workers have recently shown that it is in part the milk of hereditarily cancer-susceptible mice that enhances susceptibility to mammary carcinoma in young females they suckle.

mixed in genetic constitution as we are, it would almost surely be a terrific job to find specific resistant strains for every cancer strain. These results make one wonder, however, whether some of the successes reported from inoculations in man may not be due to specific resistant extracts having been picked up accidentally.

Apart from general resistance or susceptibility to cancer formation there are organ localization factors. Andervont and Lynch have found that di-benzanthracene in lard will cause cancer in any strain, *but* animals of susceptible strains are usually all dead before those of resistant strains begin to develop tumours, and, still more significantly, a strain susceptible to lung tumours will develop lung tumours and not necessarily tumours at the site of application of the di-benzanthracene. That looks like very strong evidence for genetic factors being primary agents in cancer. So let us quote the opposite side, that animals of strains resistant to mammary carcinoma can in some cases be made to develop mammary tumours if their young are removed at birth and they are bred very frequently. This is recent work by Halsey Bagg. But I said "often"; some animals will resist even this, and it looks as if this technique will be valuable in the selection of very highly resistant strains.

Then we may consider the fact that both in human beings and in mice tumours inside the peritoneum develop on the average at a later age than do mammary tumours. If one favours the extrinsic factor of causation one can say that the "soil is right" for tumour development at different ages in different parts of the body. If one stresses the intrinsic factor one can point out that mammary tissue matures or becomes senile earlier and that old cells do mutate more frequently, as shown by work on plants. Then there is the fact that ovaries transplanted into castrated males develop like virgin ovaries, and the animals have a cancer frequency similar to that of virgin females, which is lower than that of breeding females. I imagine that might be interpreted either way.

Perhaps I should conclude with some remarks on our own work. It is based on the working hypothesis that cancer is due to somatic cell mutation. This illustrates, of course, the peculiar weakness of the human mind and the foundation of the heredity versus environment argument. We seem to get farthest if we take one view instead of trying to see the whole problem all the time (though of course we can come up to have a look around occasionally). Well, we said: if the somatic mutation theory is right, what is the mechanism that makes some strains susceptible? One characteristic of tumours themselves is a precocious splitting of the chromosomes. This I saw in Ludford's preparations of several tumours in 1929 and again in Lewis's in 1931, since when it has become generally acknowledged. I reasoned that if this precocity should be found in non-cancerous tissues of susceptible strains and not in resistant strains it would provide a mechanism for mutation or, at any rate, be an indication of readiness to mutate. Dr. Marie Hearne and I looked at somatic cells of susceptible and resistant mice obtained from Dr. Little and thought we could see a difference in time of splitting of the chromosomes, but it was slight and difficult to be certain about. In a plant, *Fritillaria Meleagris*, I had previously found relative precocity of chromosome splitting to be correlated with failure of pairing in parts of the chromosomes. The further working hypothesis was therefore made that the chromosomes would be found to be more loosely paired at germ cell formation in susceptible than in resistant mice. The results obtained in testing this have been published in the *Canadian Journal of Research*. Suffice it to say now that we did find a striking correlation in eight strains but that two related strains were exceptional. A most heartening feature, however, is that one of these exceptional strains has since been found to be exceptional genetically also.

Dr. Hearne obtained a scholarship and went to England where she continued her work on tissue culture at Cambridge, and found that a carcinogenic agent, methylcholanthrene, causes pairing of chromosomes in body cells in tissue culture. We are now carrying on similar studies.

If we continue to get results of the same type as those of the past three years we shall be able to say that the genetic constitution is the "proximal" cause of cancer, using Rous' terminology. He can continue to claim the virus as the "primary" cause for the *specific cases* in which he can prove a virus to be present. If *we* are right, that will not lessen the importance of the extrinsic viewpoint; cancer apparently does not arise without an external cause, but if the cause is everywhere, if, as Rous says, the virus is everywhere, it is not going to be possible to do much about controlling the environmental causes; perhaps we should be looking for some resistant agency such as that of McDowell but of more general capacity.

One thing is certain; cancer results from the interaction of heredity and environment. But the only way to tackle a scientific problem is to isolate variables. This is too often forgotten. Those who are working on the extrinsic factor ought to eliminate or reduce the genetic variables by using pure strains of mice or rats of known constitution now available. It distresses a geneticist to read papers about work on

laboratory animals of unknown genetic constitution. If the experimental animals differ genetically and the treatments differ at the same time you have no idea whether the effect you get is either "primarily" or "proximally" due to heredity or environment. If you know the genetic constitution of the animals worked on, you may be able to determine whether heredity or environment is the "proximal" or "primary" cause under the particular circumstances of your experiment. We can leave aside as meaningless, or at least irrelevant, the question of which is the more "important" factor.

Association Notes

THE SIXTY-EIGHTH ANNUAL MEETING OF THE CANADIAN MEDICAL ASSOCIATION, IN CONJUNCTION WITH THE FIFTY-SEVENTH ANNUAL MEETING OF THE ONTARIO MEDICAL ASSOCIATION, TO BE HELD IN OTTAWA, ON JUNE 21, 22, 23, 24, 25, 1937

Convention Headquarters, Chateau Laurier Hotel

The Canadian Medical Association

President, H. M. ROBERTSON, Victoria;
President-elect, T. H. LEGGETT, Ottawa;
General Secretary, T. C. ROUTLEY, Toronto.

The Ontario Medical Association

President, W. K. COLBECK, Welland;
President-elect, R. K. PATERSON, Ottawa.

The Ottawa Medico-Chirurgical Society

President, R. L. GARDNER, Ottawa;
President-elect, J. H. LAPOINTE, Ottawa;
Secretary, T. L. FISHER, Ottawa.

Members of the medical profession throughout Canada will read with appreciation the subjoined message from the President-elect of the Canadian Medical Association who speaks at this time particularly on behalf of his local Society. As a Past President of this Society and as one who has always devoted himself to the best interests of his profession Dr. Leggett is highly esteemed by his Ottawa colleagues and regarded by them as being in every sense worthy to succeed those of their outstanding fellow-members who have formerly graced the position. One of these, Dr. H. B. Small, now entering upon his fifty-eighth year of practice, looks forward with unabated enthusiasm to this year's great gathering.

Message of the President-elect to the Canadian Medical Association

No doubt it is the desire of every citizen of Canada to see and have his children see the Capital City of the Dominion. The doctors of Ottawa and their wives, their sons and daughters, are enthusiastically united as hosts in attempting to present this opportunity in the most hospitable manner to the members of the Canadian Medical Association.

The young people will be welcomed by an organized program which will take care of them throughout their entire stay for the Convention, giving them the opportunity of seeing all the physical features of the Capital City as well as a goodly measure of the social side of life in the Capital.

The Ladies' Program is organized quite independently of the young people's parties, and the mothers will feel entirely free to enjoy the entertainment provided for them knowing that their families, young or full-grown, are well provided for.

Now having disposed of all the family cares and responsibilities of the members for the entire duration of their visit, these are left free to meditate and enjoy the Scientific Sessions, the Scientific Exhibits, and the Commercial Exhibits.

The Scientific Program this year has been long in course of preparation, is wide in its scope, and is outstanding.

The Scientific Exhibits will appeal strongly, as the interest of the exhibitors has all along been keen, resulting in a large and most excellent collection, attesting to the recent advances in the science of medicine.

Once a year at our Annual Meeting we greet many of our friends from coast to coast, thus ever and anon keeping up that fraternity of spirit which gladdens the heart, stores up that warmth in the inner being so necessary to real contentment, and broadens the human outlook.

For many of our profession the heavy season's work is lightening, and we are casting about for some item of mental recreation to freshen us and make us keen again for that interlude of work which we must carry on before our summer holiday begins.

Come and let the delights of these features of our Annual Meeting at Ottawa satisfy your natural desires.
May 4, 1937 (Signed) T. H. Leggett.

It is appropriate to insert here a letter received by the Committee on Arrangements from the office of His Worship, the Mayor of Ottawa.

On behalf of the Board of Control, the City Council, and the citizens of Ottawa, it gives me great pleasure to note that during the week of June 21st our City will be visited by members of the Canadian and Ontario Medical Associations.

The citizens of Ottawa will deem it a great privilege to welcome all those members of the medical profession who may be able to assemble in Ottawa at a time when the Capital City is at the height of its far-famed natural beauty.

In extending greetings we wish to include all the members of the delegates' families who happen to accompany them, being convinced that the hospitality available for them will be found to be of a most delightful kind.

(Signed) G. M. Geldert, M.D.,
May 4, 1937 Acting Mayor.

The Committee on Arrangements are thankful to say that the cordial spirit which marks the above communication is accompanied by very well-worth-while practical assistance cheerfully rendered by the City Fathers.

At their special meetings the Executives and members of Council of both major organizations expect to transact a very considerable amount of business.

On the occasion of Ontario Night (Thursday, June 24th) the members of the provincial association will hear reports of far-reaching significance. These will be embodied in summaries to be given by the General Secretary, Dr. T. C. Routley, fresh from his Old Country visit; from the President, Dr. W. K. Colbeck; and from Dr. R. K. Paterson, President-elect. The two latter have spent themselves untiringly on behalf of their constituents throughout Ontario —having travelled thousands of miles up and down the province, with the aim of securing a solid status for organized medicine. Dr. Colbeck will review, among other subjects, the relationship which now exists (temporarily only, it is hoped) between the Workmen's Compensation Board and the Drugless Practitioners.

The medical men of Ontario would do well to study the act of the Legislature which has altered the pre-existing situation. In order that their minds may be refreshed on the matter before they attend the "Ontario Night" conference, the altogether astonishing "Extract from the Bill" is reprinted below.

Extract from No. 122—BILL—THE STATUTE LAW AMENDMENT ACT, 1937. 1st Reading, March 22, 1937; 2nd Reading, March 23, 1937; 3rd Reading, March 25, 1937; Mr. Roebuck. 3rd Session, 19th Legislature, Ontario, 1 George VI, 1937.

Page 27

70. (1) Subsection 1 of Section 49 of the WORKMEN'S COMPENSATION ACT; as amended by subsection 1 of section 2 of the WORKMEN'S COMPENSATION ACT, 1932, is further amended by inserting after the words "dental aid" in the fourth line the words "the aid of drugless practitioners registered under THE DRUGLESS PRACTITIONERS ACT" so that the said subsection shall now read as follows:

(1) Every workman entitled to compensation under this Part, or who would have been so entitled had he been disabled for seven days, shall be entitled to such medical, surgical and dental aid, the aid of drugless practitioners registered under THE DRUGLESS PRACTITIONERS ACT, and hospital and skilled nursing services as may be necessary as a result of the injury, and shall be entitled to such artificial member or members and apparatus and dental appliances and apparatus as may be necessary as a result of the injury, and to have the same kept in repair for a period of one year.

(2) Subsection 2 of the said section 49, as amended by subsection 2 of section 2 of THE WORKMEN'S COMPENSATION ACT, 1932, is further amended by inserting after the words "dental aid" in the second line the words "the aid of drugless practitioners registered under THE DRUGLESS PRACTITIONERS ACT" so that the said subsection shall now read as follows:

(2) In this act "medical aid" shall mean the medical, surgical and dental aid, the aid of drugless practitioners registered under THE DRUGLESS PRACTITIONERS ACT and hospital and skilled nursing services and the artificial member or members and apparatus and repair above mentioned.

The Scientific Program is now complete and appears herewith. It may be said that, whereas in its preparation the Program Committee experienced many disappointments, it now issues in final form as representing an achievement which far excels earlier expectations. In this connection special attention is drawn to the generosity and discernment of the British Medical Association in providing for our enlightenment the services of two such eminent specialists as Mr. Watson Jones, of Liverpool, and Mr. Beckwith Whitehouse, of Birmingham, who will be among the foremost leaders in the transactions of the General Sessions. Also, to take a prominent place with them in this part of the program, as well as in the discussions of the Radiological Section, will be the famous Assistant-Director of the Radium Institute of Paris, M. Antoine Lacassagne. As a most welcome guest, this distinguished scientist will come to the Convention under the auspices of the Ottawa Medico-Chirurgical Society.

The Ladies' Committee have submitted a final program. Its preparation has involved a great deal of activity on their part and they hope that their unstinted efforts will be amply rewarded by way of such an attendance at the various social functions as will be representative of all parts of Canada. This Committee urges the necessity for incoming guests to register immediately upon their arrival at the Ladies' Headquarters in the Chateau Laurier. Each morning, committee members will be in attendance at the special registration booth to carry out arrangements for recreations of various kinds—matches, tours, swimming, tea and luncheon parties, sightseeing, etc.

SCIENTIFIC PROGRAM

Monday, June 21st

9.00 a.m.—Registration.

9.30 a.m.—Meeting of the General Council, Canadian Medical Association.

12.30 p.m.—Luncheon.

2.00 p.m.—Meeting of the General Council, Canadian Medical Association.

5.00 p.m.—Meetings of Committees.

6.00 p.m.—Meeting of Nominating Committee, Canadian Medical Association.

7.30 p.m.—Meeting of Council, Royal College of Physicians and Surgeons.

Tuesday, June 22nd

9.15 a.m.—Meeting of the General Council, Canadian Medical Association.

12.30 p.m.—Luncheon.

2.00 p.m.—Meeting of the General Council, Canadian Medical Association.

2.15 p.m.—Official opening of Exhibit Hall.

Wednesday, June 23rd

8.30 a.m.—Registration.

9.00 a.m.—General Session.

12.30 p.m.—Luncheon. Guest Speaker, His Excellency the Right Honourable Baron Tweedsmuir of Elsfield, G.C.M.G., C.H., Governor-General of Canada.

2.00 p.m.—Sectional Meetings.

8.30 p.m.—Annual General Meeting, to which all members and their ladies, guest speakers, and official delegates are invited. (Formal. Gowns and decorations in order.)
Music.

9.00 p.m.—Call to Order by the President.

- Invocation.
- Introduction of Guest Speakers and Official Delegates.
- Introduction of Senior Members and presentation of badges and certificates.
- Installation of the new President.
- Address of the President.
- Reception by the President and Mrs. Leggett, followed by dancing, cards and refreshments.

Thursday, June 24th

8.00 a.m.—Breakfast Session. Delegates from medical schools and licensing bodies to discuss intern supervision.

Thursday, June 24th—*Continued*

9.00 a.m.—Sectional Meetings.

12.30 p.m.—Luncheon.

2.00 p.m.—General Session.

2.00 p.m.—Meeting of Incoming Executive Committee.

4.00 to 6.00 p.m.—Garden Party.

7.00 p.m.—Subscription Dinner of the Ontario Medical Association, to which all members of the Canadian Medical Association and their wives are cordially invited. After the dinner there will be a short business meeting of the Ontario Medical Association followed by music and special entertainment.

Friday, June 25th

9.00 a.m.—General Session.

11.30 a.m.—Blackader Oration. Rheumatic fever and heart disease in childhood.—Dr. H. B. Cushing, Montreal.

12.00 a.m.—Luncheon. Guest Speaker, The Hon. Martin Burrell, Parliamentary Librarian, Ottawa.

Immediately following the luncheon the Canadian Medical Protective Association will hold its annual meeting.

1.00 p.m.—Annual Golf Tournament. (See special notice "Golfers Attention", May issue, p. 536.)

7.30 p.m.—Dinner, followed by—

- Presentation of prizes to all winners at the Golf Tournament.
- Special program of music and entertainment by the Gatineau Troubadours.

GENERAL SESSIONS

Wednesday, June 23rd

9.00 a.m. to 12.30 p.m.

Symposium on Jaundice:

Dr. Wm. Magner, Toronto.—Pathological aspect;

Dr. A. M. Snell, Rochester, Minn.—Medical aspect;

Dr. A. T. Bazin, Montreal.—Surgical considerations.

Mr. Beckwith Whitehouse, Birmingham, Eng.—Some impressions of a gynæcologist in post-war Europe.

Dr. R. R. Graham, Toronto.—Surgical Clinic.

Thursday, June 24th

2.00 to 4.30 p.m.

Dr. Hermann Robertson, Victoria, B.C.—Valedictory Address of the Past President.

Dr. B. P. Watson, New York.—Post-partum sepsis.

Dr. Antoine Lacassagne, Paris.—The relation between hormones and cancer.

Dr. A. H. Gordon, Montreal.—Medical Clinic.

Dr. G. M. Geldert, Ottawa.—Demonstration of the electrical stethoscope.

Friday, June 25th

9.00 a.m. to 12.30 p.m.

Mr. Watson Jones, Liverpool.—Modern developments of fracture treatment. (Illustrated with films).

Dr. A. T. Mathers, Winnipeg.—Psychological aspects of medicine.

Dr. H. B. Cushing, Montreal.—Blackader Oration. Rheumatic fever and heart disease in childhood.

SECTIONAL MEETINGS

Wednesday afternoon and Thursday morning have been set aside for Sectional Meetings.

The following is a list of the speakers and subjects in each Section:

Section of Anæsthesia

WEDNESDAY, JUNE 23RD

2.00 p.m.—

Anæsthesia in children.—Dr. C. H. Robson, Toronto.

The present unusual opportunities for medical men in anæsthesia.—Dr. John S. Lundy, Rochester, Minn.

Anæsthesia from the patient's point of view.—Dr. Harold Griffith, Montreal.

Carbon dioxide.—Dr. Ralph Waters, Madison, Wis.

Anæsthetic procedures as standardized for certain types of operations in a large general hospital.—Dr. H. J. Shields, Toronto.

Section of Dermatology

WEDNESDAY, JUNE 23RD

2.00 p.m.—

The treatment of recurrent erythema multiforme.—Dr. Norman Wrong, Toronto.

Roentgen and ultra-violet radiation in dermatology.—Dr. D. E. H. Cleveland, Vancouver.

Facts, fads and fancies in the treatment of acne vulgaris.—Dr. L. P. Ereaux, Montreal.

Disturbances of the sebaceous glands.—Dr. W. R. Jaffrey, Hamilton.

Wednesday, June 23rd—*Continued*

The past year at the skin clinic, Hospital for Sick Children, Toronto.—Dr. H. A. Dixon, Toronto.

THURSDAY, JUNE 24TH

9.00 a.m.—

Kaposi's varicelliform eruption. Report of a case with experimental evidence in support of a virus etiology.—Dr. Geo. S. Williamson, Ottawa.

Urticaria.—Dr. Harold Orr, Edmonton.

A dermatologist reviews his private practice.—Dr. E. J. Trow, Toronto.

Contact eczema from dyed clothing.—Dr. J. F. Burgess, Montreal.

Unusual skin malignancies, diagnosis and treatment.—Dr. B. W. MacDonald, Ottawa.

Kraurosis and leukoplakia vulvæ and pruritus with lichenification treated by perineal sensory nerve resection.—Dr. B. Usher and Dr. A. D. Campbell, Montreal.

Section of Medicine

WEDNESDAY, JUNE 23RD

2.00 p.m.—

Gastro-intestinal hæmorrhage.—Dr. H. A. Cave, London.

Discussion to be opened by Dr. Albert LeSage, Montreal and Dr. H. A. Peacock, Hamilton.

Metastasis and metastases.—Dr. E. L. Pope, Edmonton.

Hyperpiesia.—Dr. K. A. MacKenzie, Halifax.

Discussion to be opened by Dr. J. W. Tice, Hamilton.

Clinical aspects of precordial pain.—Dr. W. Ford Connell, Kingston.

Discussion to be opened by Dr. R. S. Stevens, Ottawa.

Diagnosis of some major vascular accidents.—Dr. D. M. Baltzan, Saskatoon.

Discussion to be opened by Dr. F. C. Neal, Peterborough.

THURSDAY, JUNE 24TH

9.00 a.m.—

An interpretation of some mechanisms in the menstrual cycle and in pregnancy in the light of sex-hormone determinations.—Dr. John S. L. Browne, Montreal.

Recent immunological and pathological studies in experimental poliomyelitis and their significance. — Dr. Maurice Brodie, New York.

Discussion to be opened by Dr. Fred MacKay, Montreal.

The treatment of tuberculosis after forty years. Dr. Jules Prevost, Montreal.

Thursday, June 24th—*Continued*

The abolition of clinical tuberculosis by anticipation and control; a definite scheme and its application.—Dr. W. E. Ogden, Toronto.
Discussion to be opened by Dr. H. A. Farris, Saint John and Dr. G. J. Wherrett, Ottawa.

X-ray therapy in bronchial asthma.—Dr. J. M. McEachern, Winnipeg.

Section of Obstetrics and Gynæcology

WEDNESDAY, JUNE 23RD

2.00 p.m.—

The endocrine factors in normal and abnormal menstruation. — Dr. Melville C. Watson, Toronto.
Discussion to be opened by Dr. A. D. Campbell, Montreal.

Sterility.—Dr. Ernest Couture, Ottawa.
Discussion to be opened by Dr. A. B. Whytock, Niagara Falls, and Dr. H. Dover, Ottawa.

Menorrhagia of the menopause.—Dr. J. D. McQueen, Winnipeg.
Discussion to be opened by Dr. E. V. Shute, London and Dr. B. P. Watson, New York.

Obstetrical complications. — Dr. John Mann, Toronto.
Discussion to be opened by Dr. W. J. Stevens, Ottawa and Dr. N. W. Philpott, Montreal.

Endometriosis.—Dr. Geo. Hooper, Ottawa.

THURSDAY, JUNE 24TH

9.00 a.m.—

Pre-eclamptic toxæmia. — Dr. W. P. Tew, London.
Discussion to be opened by Dr. P. A. McLeod, Kingston.

Abortion.—Dr. H. C. Burgess, Montreal.
Discussion to be opened by Dr. Norman Guiou, Ottawa and Dr. O. W. Niemeier, Hamilton.

Pre-natal and post-natal care.—Dr. H. B. Atlee, Halifax.
Discussion to be opened by Dr. D. G. McIlwraith, Hamilton.

Treatment of genital prolapse.—Dr. Wm. Scott, Toronto.
Discussion to be opened by Dr. G. W. Mylks, Kingston.

Salpingitis.—Mr. Beckwith Whitehouse, Birmingham, Eng.

Section of Ophthalmology

WEDNESDAY, JUNE 23RD

2.30 p.m.—

Business Session.

The etiology and treatment of keratitis neuroparalytica.—Dr. J. A. MacMillan and Dr. W. V. Cone, Montreal.

Wednesday, June 23rd—*Continued*

Recurrent styes — treatment by staphylococcus toxoid. — Dr. Alexander E. MacDonald, Toronto.

Experiences in and the treatment of detachment of the retina.—Dr. Wm. Lowry, Toronto.

THURSDAY, JUNE 24TH

9.30 a.m.—

Lighting as a more exact science.—Dr. G. Stuart Ramsey, Montreal.

Survey of cataract operative procedures in the last thirty years.—Dr. Charles O'Connor, Kingston.

The ophthalmoscopic findings in one hundred consecutive cases of cardiovascular hypertension.—Dr. F. T. Tooke and Dr. John Nicholls, Montreal.

Section of Oto-Laryngology

WEDNESDAY, JUNE 23RD

2.00 p.m.—

Symposium.—Naso-genital relationship and the treatment of atrophic rhinitis with œstrogenic substances.

- Dr. Hector Mortimer, Montreal.—The constitutional factor in atrophic rhinitis: its probable nature and etiological significance;
- Dr. J. B. Collip, Montreal.—The biochemical aspect;
- Dr. R. P. Wright, Montreal.—The clinical aspect.

Discussion to be opened by Dr. H. S. Birkett, Montreal.

THURSDAY, JUNE 24TH

9.00 a.m.—

Differential diagnosis of upper dysphagia.—Dr. A. H. Veitch and Dr. E. H. Shannon, Toronto.
Discussion to be opened by Dr. Perry Goldsmith, Toronto.

Stricture of the œsophagus.—Dr. G. E. Hodge and Dr. E. C. Scharfe, Montreal.
Discussion to be opened by Dr. D. Ballon, Montreal.

Three cases of petrositis.—Dr. D. E. S. Wishart, Toronto.
Discussion to be opened by Dr. J. K. M. Dickie, Ottawa.

Functional testing of the ear.—Dr. W. J. McNally, Montreal.
Discussion to be opened by Dr. R. S. Pentecost, Toronto.

Section of Pædiatrics

WEDNESDAY, JUNE 23RD

2.00 p.m.—

Childhood tuberculosis.—Dr. A. M. Jeffrey, Ottawa.

Discussion to be opened by Dr. W. E. Ogden, Toronto and Dr. H. Wright, Montreal.

Rubella or German measles.—Dr. H. B. Cushing, Montreal.

Discussion to be opened by Dr. L. MacHaffie, Ottawa and Dr. W. T. Shirreff, Ottawa.

Cerebral pneumography in childhood.—Dr. A. E. Childe, Montreal.

Discussion to be opened by Dr. A. H. Rolph, Toronto.

The physiology of the central nervous system in regard to pædiatrics.—Dr. Howard Spohn, Vancouver.

The treatment of epilepsy in children.—Dr. H. M. Keith, Montreal.

Discussion to be opened by Dr. C. K. Rowan-Legge, Ottawa.

THURSDAY, JUNE 24TH

9.00 a.m.—

Immunization in childhood.—Dr. Donald Fraser, Toronto.

Discussion to be opened by Dr. Theo Lomer, Ottawa.

The obstructed or mechanical appendix.—Dr. Alan Brown and Dr. D. E. Robertson, Toronto.

The clinical use of para-amino-benzene-sulphonamide or its derivatives in the treatment of beta hæmolytic streptococcal infection.—Dr. Perry Long, Baltimore, Md.

Discussion to be opened by Dr. Patrick Maloney, Ottawa and Dr. R. Struthers, Montreal.

The normal occurrence of fetal respiration and its relation to the abnormalities of the newborn.—Dr. F. S. Snyder, Baltimore, Md.

Discussion to be opened by Dr. W. Stevens, Ottawa and Dr. B. P. Watson, New York.

Sudden death in young infants.—Dr. Alton Goldbloom and Dr. F. W. Wiglesworth, Montreal.

The present status of vitamin therapy in childhood.—Dr. F. F. Tisdall, Toronto.

Section of Radiology

WEDNESDAY, JUNE 23RD

2.00 p.m.—

Diverticulitis of the sigmoid colon; a radiological study.—Dr. A. C. Singleton and Dr. M. R. Hall, Toronto.

Benign conditions of the uterus.—Dr. J. E. Gendreau, Montreal.

Wednesday, June 23rd—*Continued*

(Subject to be announced).—Dr. Antoine Lacassagne, Paris.

Radiological education.—Dr. W. A. Jones, Kingston.

The radiologist, an integral part in the scheme of things medical.—Dr. W. H. McGuffin, Calgary.

THURSDAY, JUNE 24TH

9.00 a.m.—

Diagnosis in the chest.—Dr. H. R. Corbett, Kentville.

Malignant disease of the breast.—Dr. G. E. Richards and Dr. A. D. Irvine, Toronto.

X-ray treatment of non-malignant skin conditions.—Dr. C. M. Henry, Regina.

Urology.—Dr. W. L. Ritchie, Montreal.

Differential diagnosis of bone tumours.—Dr. Bernard Mooney, Edmonton.

Rheumatic Diseases

WEDNESDAY, JUNE 23RD

2.00 p.m.—

Some problems of rheumatic diseases.—Dr. A. A. Fletcher, Toronto.

The present aspects of rheumatic diseases.—Dr. Albert LeSage, Montreal.

Canadian organization to combat rheumatic diseases.—Dr. W. S. Barnhart, Ottawa.

Report of a Canadian survey of rheumatic diseases.—Dr. F. S. Burke, Ottawa.

THURSDAY, JUNE 24TH

9.00 a.m.—

Differential diagnosis and treatment of chronic arthritis.—Dr. G. Douglas Taylor, Montreal.

Why not a status in Canada for gold therapy of arthritis?—Dr. A. W. Bagnall, Vancouver.

Business Session of the Canadian Rheumatic Disease Association.

Section of Surgery

WEDNESDAY, JUNE 23RD

2.00 p.m.—

Certain aspects of the treatment of fractures of the forearm.—Dr. G. W. Armstrong, Ottawa.

Discussion to be opened by Dr. Frank O. Patterson, Vancouver and Dr. Fraser B. Gurd, Montreal.

Fractures of the skull.—Dr. Arthur Elvidge, Montreal.

Discussion to be opened by Dr. Geo. A. Ramsay, London and Dr. K. G. McKenzie, Toronto.

Wednesday, June 23rd—*Continued*

Automobile injuries to the head.—Dr. Fulton Risdon, Toronto.

Discussion to be opened by Dr. B. F. McNaughton, Montreal and Dr. W. O. Stevenson, Hamilton.

Important minor points in local anæsthesia.—Dr. Philip D. Woodbridge, Boston, Mass.

Discussion to be opened by Dr. P. H. T. Thorlakson, Winnipeg and Dr. John S. Lundy, Rochester, Minn.

Pruritus ani.—Dr. J. T. Danis, Toronto.

Discussion to be opened by Dr. C. K. P. Henry, Montreal and Dr. Maxwell Yelland, Peterborough.

THURSDAY, JUNE 24TH

9.00 a.m.—

Infections and injuries in the hands.—Dr. J. H. Couch, Toronto.

Discussion to be opened by Dr. L. H. McKim, Montreal and Dr. A. T. Gillespie, Fort William.

The treatment of bone and joint infections (illustrated with films).—Mr. Watson Jones, Liverpool, Eng.

Some post-operative complications following abdominal operations.—Dr. Neil J. Maclean, Winnipeg.

Discussion to be opened by Dr. J. K. McGregor, Hamilton and Dr. L. J. Austin, Kingston.

An outline of the indications for the operation of pulmonary lobectomy: some remarks upon the choice of suitable patients for the operation and the results that may be expected.—Dr. R. M. Janes, Toronto.

Discussion to be opened by Dr. E. W. Archibald, Montreal and Dr. D. A. Carmichael, Ottawa.

The clinical problem of the nodular breast.—Dr. Fulton Gillespie, Edmonton.

Discussion to be opened by Dr. Jas. Miller, Kingston and Dr. Jas. C. Armour, Montreal.

Section of Urology

WEDNESDAY, JUNE 23RD

2.00 p.m.—

Hæmaturia in general practice.—Dr. R. A. McComb, Toronto.

Conservative treatment in urogenital tuberculosis.—Dr. R. C. Hastings, Quebec.

Genito-urinary tuberculosis.—Dr. J. C. McClelland and Dr. K. F. Davis, Toronto.

Wednesday, June 23rd—*Continued*

Lesions of the bladder neck and trigone in the female.—Dr. David W. MacKenzie, Montreal.

Neoplasms of the renal capsule.—Dr. W. J. Deadman and Dr. S. F. Penny, Hamilton.

THURSDAY, JUNE 24TH

9.00 a.m.—

Hyperparathyroidism,—diagnosis and treatment with special reference to kidney complications.—Dr. A. C. Abbott, Winnipeg.

The so-called silent renal stone.—Dr. E. D. Busby, London.

Late results of the treatment of bladder tumours.—Dr. Gordon S. Foulds, Toronto.

Tumours of the ureter.—Dr. Frank S. Patch, Montreal.

The significance of pain in the upper urinary tract.—Dr. Robin Pearse, Toronto.

Supra-pubic prostatectomy, methods and results; a comparison with transurethral resections.—Dr. R. E. Powell, Montreal.

Section of Historical Medicine

The Historical Section has arranged with the Dominion Archives to have on display many books, documents, and pictures relating to the History of Medicine in Canada. Members of the staff, who are familiar with various items will be present to explain the material. Some of the collection of Dr. H. B. Small, Historian for the Ottawa Medico-Chirurgical Society, will also be shown.

Members of the Canadian Medical Association, their wives and friends, are cordially invited to visit the Dominion Archives, which is about five minutes drive from the Chateau Laurier, between the hours of 9 a.m. and 5 p.m.

UNIVERSITY OF TORONTO ALUMNI—ATTENTION!

A Coronation Year Alumni Dinner has been arranged under the direction of the President, Dr. R. E. Wodehouse, for Wednesday Evening, June 23rd, at 6.30 p.m., at the Rideau Club, during the annual meeting of the Canadian Medical Association in Ottawa.

Members should obtain their dinner tickets from the Canadian Medical Association registration desk and, when doing so, should inform the clerk of their class year, in order that seating arrangements may be made accordingly.

It is your Dinner—informal—no speaker—and will not conflict with sessional meetings.

Don't miss this opportunity of spending a happy hour reminiscing with old college pals. THE DATE IS JUNE 23rd.

LOCAL PROGRAM COMMITTEES

General Sessions

Chairman—Dr. H. B. Moffatt
Secretary—Dr. J. E. Plunkett

Dr. S. Evans, Dr. Norman Guiou, Dr. Geo. S. MacCarthy, Dr. Geo. Armstrong, Dr. C. H. Brown, Dr. J. B. Thackeray, Dr. J. H. Lapointe, Dr. A. McNabb, Dr. C. Laidlaw, Dr. W. S. Lyman.

Section of Medicine

Chairman—Dr. H. Laidlaw
Secretary—Dr. R. A. Starrs

Dr. W. S. Lyman, Dr. J. E. Plunkett, Dr. S. Mirsky, Dr. Wm. Guest, Dr. L. Brown, Dr. S. Service.

Section of Surgery

Chairman—Dr. Geo. S. MacCarthy
Secretary—Dr. G. W. Dunning

Dr. F. P. Quinn, Dr. T. H. Leggett, Dr. H. B. Moffatt, Dr. R. K. Paterson, Dr. C. H. Brown, Dr. Geo. Hooper, Dr. R. E. Valin, Dr. S. Evans.

Section of Obstetrics and Gynæcology

Chairman—Dr. Geo. Hooper
Secretary—Dr. G. W. B. York

Dr. E. Couture, Dr. H. Dover, Dr. J. F. Puddicombe.

Section of Oto-Laryngology

Chairman—Dr. J. K. M. Dickie
Secretary—Dr. J. R. Delahay

Dr. W. P. E. Paterson, Dr. J. T. Coupal.

Section of Ophthalmology

Chairman—Dr. W. G. Fraser
Secretary—Dr. J. T. Coupal

Section of Radiology

Chairman—Dr. Paul Brodeur
Secretary—Dr. A. McNabb

Dr. R. K. Paterson.

Section of Dermatology

Chairman—Dr. G. S. Williamson
Secretary—Dr. B. W. MacDonald

Dr. S. E. Grimes.

Section of Pædiatrics

Chairman—Dr. Geo. Campbell
Secretary—Dr. J. C. Armstrong

Section of Urology

Chairman—Dr. Wm. Hutchinson
Secretary—Dr. E. Gaulin

Section of Anæsthesia

Chairman—Dr. G. M. Geldert
Secretary—Dr. H. Burgess

Dr. H. T. Jost, Dr. N. Sauve, Dr. L. E. Roche.

Section of Rheumatic Diseases

Chairman—Dr. W. S. Lyman
Secretary—Dr. W. S. Barnhart

Section of Historical Medicine

Chairman—Dr. S. Mirsky
Secretary—Dr. J. J. Heagerty

Committee on Arrangements

Chairman—Dr. R. E. Valin
Secretary—Dr. T. L. Fisher

Drs. J. H. Alford, C. H. Brown, Geo. Campbell, A. P. Davies, C. G. Gunn, D. A. Whitton, D. A. Carmichael, G. M. Geldert, J. L. Lamy, P. Belanger, J. F. Argue, N. M. Harris, W. Hutchinson, G. S. MacCarthy, S. M. Nagle, F. P. Quinn, W. J. Stevens, J. H. Lapointe, C. Laidlaw, J. M. Laframboise, R. L. Gardner, J. L. Coupal, W. S. Lyman.

SUB-COMMITTEES OF COMMITTEE ON ARRANGEMENTS

Commercial Exhibits

Chairman—Dr. Geo. Armstrong

Dr. G. W. B. York, Dr. W. A. Guest, Dr. E. P. Byrne, Dr. C. T. Fink

Committee on Housing

Chairman—Dr. J. H. Alford

Dr. R. Law, Dr. Wm. E. Caven, Dr. Hector Featherstone, Dr. L. W. Brown, Dr. F. S. Burke.

Committee on Publicity

Chairman—Dr. Campbell Laidlaw

Dr. G. M. Geldert, Dr. F. W. C. Mohr, Dr. E. Couture, Dr. G. O. Barclay.

Committee on Scientific Exhibits

Chairman—Dr. G. S. Williamson

Dr. Jos. Gaulin, Dr. W. G. Watt, Dr. S. E. Grimes Dr. H. L. Sims.

Committee on Registration

Chairman—Dr. S. L. E. Danby

Dr. J. H. Burgess, Dr. J. C. Humphreys, Dr. P. B. Belanger, Dr. A. V. Kniewasser, Dr. E. Smith, Dr. L. L. Derby, Dr. T. J. Scobie.

Golf Committee

Chairman—Dr. Wm. Hutchinson

Dr. L. E. Roche, Dr. A. P. Davies, Dr. R. Law.

Junior Committee

Chairman—Dr. F. W. Jeffrey

Dr. J. F. Puddicombe, Dr. L. J. Walters, Dr. Wallace Troup, Dr. L. P. Little, Dr. L. D. Wilson, Dr. R. R. Barnes, Dr. C. K. Rowan-Legge.

Committee on Badges

Chairman—Dr. D. T. Burke

Dr. J. H. Hamlin, Dr. W. J. Gordon, Dr. K. C. Butler, Dr. G. E. Grondin.

Committee on Entertainment

Chairman—Dr. J. M. Laframboise

Dr. C. G. Gunn, Dr. W. W. Mackay, Dr. C. F. Williams.

Committee on Transportation

Chairman—Dr. W. P. E. Paterson

Dr. H. E. Hanna, Dr. S. F. Service, Dr. J. V. Berry, Dr. H. R. T. Mount, Dr. C. W. Kelley, Dr. H. H. Gordon.

CENTRE BLOCK, PARLIAMENT BUILDINGS, OTTAWA

THE LADIES' PROGRAM

MONDAY, JUNE 21ST

Morning

Sight-seeing.—Visits to: Archives, Royal Mint, Houses of Parliament, Filtration Plant.

Recreation.—Golf, tennis, swimming, riding.

Afternoon

1.00—Luncheon at Country Club; hostess, Mrs. T. H. Leggett. For the wives of the Councillors.

TUESDAY, JUNE 22ND

Morning

Recreation.—9.30, Special drive; 11.00 to 1.00, morning tea at Longfields, Aylmer Rd.; hostess, Mrs. J. F. Kidd.

Evening

7.15—Ladies' dinner, Chateau Laurier; hostess, Ottawa Doctors' Wives' Association. For the wives of Councillors.

WEDNESDAY, JUNE 23RD

Morning

Recreation.

Sight-seeing.—11.00: Drive about the city.

Afternoon

1.00—Luncheon at golf clubs.

Evening

8.30—Ceremonial, Canadian Medical Association, Chateau Laurier. President's Reception and Dance.

THURSDAY, JUNE 24TH

Morning

Recreation. — Sight-seeing. Musical ride, R.C.M.P., Rockcliffe.

Afternoon

4.30—Garden Party at Country Club; host, The Ottawa Medico-Chirurgical Society.

Evening

7.15—Banquet, Chateau Laurier; under the auspices of the Ontario Medical Association.

FRIDAY, JUNE 25TH

Morning

Recreation.—Sight-seeing.

Afternoon

4.00—Terrace Tea, Chateau Laurier.

The General Conveners and the Conveners of Special Committees who have drawn up the above program are designated together with their General Secretary as "The Ladies' Convention Council", and are as follows.

General Conveners.—Mrs. T. H. Leggett, Mrs. R. K. Paterson.

Secretary.—Mrs. H. B. Moffatt.

Conveners of Special Committees

Registration and Information.—Mrs. H. C. Church, Mrs. G. S. McCarthy.

Reception.—Mrs. J. F. Kidd, Mrs. F. W. C. Mohr.

Transportation.—Mrs. George Hooper, Mrs. George Campbell.

Recreation.—Ladies, Mrs. F. W. McKinnon, Mrs. W. G. Fraser;
Young People, Mrs. R. Lorne Gardner.

Social.—Mrs. R. E. Valin.

Publicity.—Mrs. C. A. Young.

Financial.—Mrs. George Williamson.

JUNIOR PROGRAM

What may be expected in the way of entertainment facilities for young people can be gathered from the report given hereunder recently submitted by the Convener of the Young People's Committee, Dr. Fred W. Jeffrey.

Final arrangements have been completed for the entertainment of all young people coming to the Convention in June. It is hoped the doctors will take the opportunity of bringing their children to the Capital where they will be entertained throughout their entire stay, as demonstrated by the following program.

WEDNESDAY

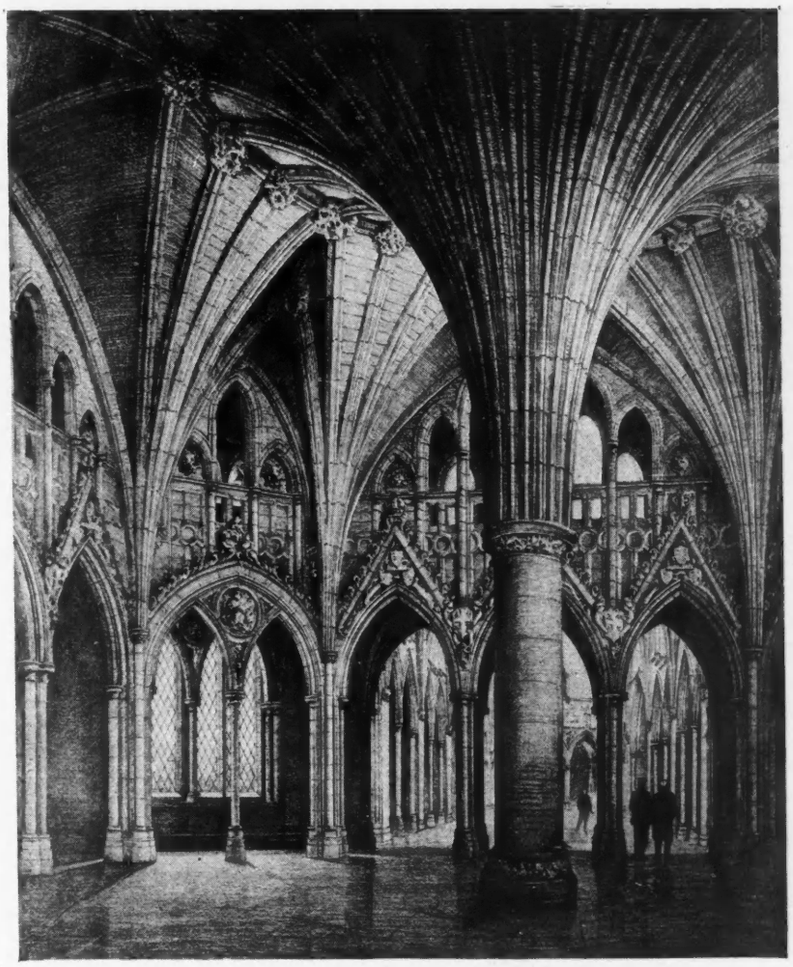
a.m.—Registration.
Noon.—Luncheon at Chateau Laurier.
2.00 p.m.—Sight-seeing tour of city.
4.00 p.m.—Afternoon tea at Mrs. Laframboise's summer home.
9.00 p.m.—Canadian Medical Association Reception and Dance.

THURSDAY

a.m.—Golf, tennis, riding and swimming.
2.00 p.m.—Boat trip up Rideau river.
5.00 p.m.—Garden party at Country Club.
9.30 p.m.—Young People's Dance at Royal Ottawa Golf Club.

FRIDAY

10.30 a.m.—Swimming party and exhibition of diving and swimming at Chateau Laurier pool.
Noon.—Luncheon at Chateau Laurier.
2.00 p.m.—Tour of Mint, Archives and Parliament Buildings.
5.00 p.m.—Tea on Chateau Laurier Terrace.
Organized golf, tennis, riding and swimming at all times.



CONFEDERATION HALL, PARLIAMENT BUILDINGS, OTTAWA

The young people coming to Ottawa are requested to bring their tennis racquets and golf clubs, and it is hoped that they will register promptly on their arrival in Ottawa at the Young People's Registration Booth in the Chateau Laurier.

Scientific Exhibitors and Exhibits

The Chairman of the Committee on Scientific Exhibits, Dr. George S. Williamson, in having completed an undertaking notable in the annals of scientific demonstration in this country, has handed in a list of Exhibitors and Exhibits as follows.

Dr. M. C. Watson, Toronto. "The causes of Normal and Abnormal Hæmorrhage from the Uterus."

A series of trans-illuminated gross and microscopic sections with accompanying photographs in black and white and in colour, together with suitable charts, will be arranged to illustrate:—(a) The uterus with completely developed endometrium previous to menstruation. (b) The uterus showing endometrium during menstruation. (c) The endometrium under continuous influence of œstrin. (d) The endometrium under continuous influence of progestin. (e) Pathological conditions of uterus and cervix which cause bleeding. Sections or photographs of the condition in the ovary responsible for (a), (b), (c) and (d) will be shown where possible.

Drs. H. Oertel, T. R. Waugh, W. H. Chase and J. E. Pritchard, Pathological Institute, McGill University. (a) "Pathological Lesions in Bones and Joints." (b) "The Histogenesis and Classification of Primary Tumours of Bones with Special Reference to Osteogenetic Tumours."

(a) About 65 selected wet and dry mounts of various bone and joint lesions with about 12 pictures. Fifteen lantern slides in individual mounts. A complete skeleton of Paget's Disease mounted in glass case. (b) A classification of bone tumours, charts, roentgenograms and photographic illustrations of the development of bone in relation to tumours, and the various types of tumours as set forth in the classification. Mounted gross museum specimens representing tumour types will also be displayed.

Department of Pensions and National Health, Laboratory of Hygiene, Ottawa. (a) "Physiological Standardization of Drugs." (b) "Modification of Smith Fermentation Tube."

International and Canadian Standards of Drugs used for Physiological Testing, together with charts, tables and photographs explaining the methods of tests and indicating some results of the standardization of drugs in Canada.

Dr. W. S. Stanbury and Miss Lucile Howell, Mountain Sanatorium, Hamilton. "The Laboratory Diagnosis of Intrathoracic Tumours."

Pathological specimens photograph and photomicrograph transparencies of intrathoracic tumours diagnosed by various biopsy methods. Techniques for the demonstration of tumour cells in serious effusions and in material removed through the bronchoscope or by means of aspiration biopsy are adequately illustrated. Representative case histories with transparencies of roentgenograms are included. The exhibit will serve to call attention to a variety of neoplastic processes confused clinically with pulmonary tuberculosis, in which laboratory methods may be of assistance in clarifying the diagnosis.

Dr. J. H. Elliott, Academy of Medicine, Toronto and Dr. W. S. Stanbury, Mountain Sanatorium, Hamilton. "The History of Tuberculosis."

Early and late editions of the works of pioneer investigators in the study of tuberculosis and diseases of the chest, supplemented by portraits and photographs prepared as transparencies. The exhibit also includes photographs of early sanatoria and hospitals for tuberculosis.

Dr. L. W. Thompson, Toronto Hospital for Consumptives, Weston, Ont. "Some Points in the Diagnosis and Treatment of Pulmonary Tuberculosis."

X-rays and case histories will be presented to show: (a) The necessity for close and prolonged follow-up of adolescent and young adult contacts, even though the first x-ray is clear. (b) Pulmonary tuberculosis may advance to cavitation even though temperature and pulse are normal and there are no abnormal physical signs in the chest. (c) Gain in weight and normal temperature and decreased sputum are not absolute indications of healing. (d) Rapidity with which cavitation can develop. (e) Application and value of collapse therapy, such as pneumothorax and thoracoplasty, with a comparison to the result obtained by bed rest only. (f) Clear x-ray at the time of hæmoptysis but parenchymal shadows developing later.

Drs. A. M. Snell and D. L. Wilbur, Mayo Foundation, Rochester. "Deficiency States Due to Disease in the Gastro-Intestinal Tract."

Histories, clinical data, photographs, and roentgenograms of twelve selected cases with definite abnormality of the digestive tract, illustrating the importance of these abnormalities in the production of nutritional deficiency states. These are shown by means of large illuminated boxes in the form of transparencies.

Dr. J. N. Petersen, Montreal Neurological Institute of McGill University. "Roentgenographic and Photographic."

X-ray films showing normal and pathological findings. The series includes films of the skull and vertebral column, of the intracranial and intra-spinal contents as outlined by air injections and opaque media, of normal and pathological calcification within the brain, etc. The photographic exhibit shows the technique and set-up used in the Operating Room of the Montreal Neurological Institute, and includes photographs taken during operations on the brain, spinal cord, and peripheral nerves.

Dr. M. C. Morrison, London, Ont. "Cholecystography."

Transparencies outlining various methods of x-ray examination of the gall bladder with interpretation of findings. Radiographs with legends demonstrating various types of normal and pathological gall bladders with surgical findings. Tabulation of results of oral cholecystography and consideration of the causes of error in deductions. Specimens of various forms of gallstones will be exhibited, with radiographic demonstration of variations in the density of stones and the necessity for careful technique for adequate visualization.

Drs. A. Frappier and J. A. Baudoin, University of Montreal. "Experimental and Statistical Work on BCG Vaccination at the University of Montreal."

(a) Mounted specimens of vaccinated and unvaccinated guinea pigs which have been inoculated with virulent tubercle bacilli. The resistance of the BCG-vaccinated guinea pigs to the virulent infection is easily appreciated by comparing the microscopical lesions found in the mounted animals and on the spleens of about 25 animals of each group used in the above experiment. (b) Mounted organs of guinea pigs showing lesions attributable to BCG. (c) Apparatus used in the preparation of BCG. (d) Live guinea pigs vaccinated two years ago with BCG.

Exhibit on statistical work consists of: (a) The distribution of BCG in Canada and Newfoundland by the University of Montreal. (b) Graphs and tables showing the reduction of the general mortality rate and of the mortality rate by tuberculosis in children vaccinated with BCG. (c) The technique of vaccination and re-vaccination by BCG. (d) Complete bibliography covering BCG. A substantial notice concerning BCG vaccination will be distributed.

Drs. J. S. Lundy, L. H. Mousel, E. B. Tuohy and R. C. Adams, Mayo Foundation, Rochester. "Technique of Block Anæsthesia."

Two life-sized models to demonstrate various types of block anæsthesia such as paravertebral, brachial plexus, intercostal, spinal, sacral, etc. Important

superficial landmarks of approach as well as anatomical points where the solution is deposited are demonstrated. Explanatory legends around the model describe each technique.

Dr. A. Marin, Montreal. "Moulages of Skin Diseases."

Approximately 100 moulages of a representative collection of skin diseases.

Dr. E. A. Linell, Banting Institute, University of Toronto. "Neuropathology."

(a) A series of museum specimens of cerebral tumours. (b) A series of museum specimens of cerebral hæmorrhages. (c) Wallerian degeneration: A series of photomicrographic prints.

drawings of the technique of lobectomy as described by Drs. Shenstone and Janes. Perhaps some specimens of tourniquet for lobectomy and, if possible, some wax models showing certain stages in the operations. Photomicrographs (enlarged) showing pathology of bronchiectasis.

Dr. P. Poirier, Montreal. "The Landmark of Late Heredosyphilis."

Thirty photographs representing clinical manifestations of late heredosyphilis.

Dr. L. Gross, The Mount Sinai Hospital, New York. "Experimental Studies on the Blood Supply to the Heart in Relation to Coronary Occlusion."

Charts, drawings, photographs and roentgenograms

MEMORIAL CHAMBER, PARLIAMENT BUILDINGS, OTTAWA

Dr. P. H. Thorlakson, Winnipeg. "Gastroscopy."

A photographic display of the progress in the technique of gastroscopy, the instruments at present in popular use, x-rays of gastric lesions, coloured reproductions of the normal stomach and pathological specimens removed surgically. Also plates indicating the indications and contraindications for gastroscopy and demonstration of the technique.

Dr. Robert M. Janes, Toronto. "Surgery of the Chest."

A group of pathological specimens illustrating the place of lobectomy in surgery of the chest; also, x-rays illustrating the pictures obtained following lipiodol injection and various types of bronchiectasis. Artists'

illustrating the mechanism by which the heart compensates for coronary narrowing. Illustrations of results of experimental attempts to enrich the myocardial vascular nutrition. Results of physiological studies on experimental coronary occlusion under various conditions.

Dr. J. M. Murray, Ottawa. "Reconstructive Surgery."

1. Ruptured external lateral ligament of knee. (a) Motion picture before operation. (b) Motion picture after operation.

2. Bony anklyosis of the elbow. (a) Motion pictures before operation, showing patient and x-ray of elbow. (b) Motion picture after arthroplasty using fascia lata.

3. Traumatic flail elbow. (a) Motion pictures showing patient before operation. (b) Motion pictures

of operative procedure. (c) Patient after operation showing end results.

4. Original work using skeleteal traction for correction of lateral curvatures of the spine (scoliosis).

5. Bone specimens of tuberculous knees and spines, before and after the Hibbs fusion operation.

6. Specimen of spine of lumbar sacral region demonstrating causes of low back pains, sciatica, etc.

7. Intra-capsular fractures of hips treated by blind nailing with a Smith Peterson nail. X-rays before and after, also motion pictures.

Dr. J. Miller, Queen's University, Kingston. "Series of Aneurysms of Aorta and Heart."

A series of aneurysms of the thoracic and abdominal aorta will be shown demonstrating the various situations where they occur, their mode of extension, and the channels they press upon and through which they rupture.

A series of aneurysms of the heart will be shown including one case of multiple small aneurysms of the coronary artery in a case of periarteritis nodosa.

Drs. L. J. Rhea, I. M. Rabinowitch and A. T. Bazin, Montreal General Hospital. "Jaundice."

Charts and slides illustrating diagrammatically:—(a) Physiology of bile formation and excretion. (b) Hæmolytic jaundice—types, tests, diagnosis, treatment. (c) Obstructive jaundice—types, sites, pathological changes, diagnosis, treatment. (d) Jaundice due to stone—sites, effects. (e) Jaundice due to malignancy—sites, effects. (f) Slides in illuminated box, showing pathological lesions causing jaundice.

Drs. J. W. Tice and H. R. McAlister, Hamilton General Hospital. "Diabetic Exhibit."

(a) Charts illustrating the use of protamine insulin and protamine zinc insulin for periods up to 12 months. (b) Exhibit of serial photographs showing the treatment of foot and other infections in diabetes. (c) Wax models of feet showing various degrees of gangrene encountered in diabetes. (d) Exercise studies. The effect of rowing and rugby football on the blood sugar and urine of athletes. Charts show results before and after competition. (e) Use of 6 meal diets with protamine insulin.

Dr. T. W. M. Cameron, Institute of Parasitology, McGill University. "Parasitic Worms of Man in Canada."

Demonstration of roundworms, and tapeworms of man, and Trematodes of animals which may live in man, including "Swimmers' itch". The exhibit will cover the identification, life-cycle, prevention and pathology of the important forms, and will be presented in the form of models, specimens and photographs.

Dr. D. Taylor, Royal Victoria Hospital, Montreal. "X-rays of Various Types of Chronic Arthritis."

Drs. W. A. Jones and R. C. Burr, Queen's University, Kingston. "A Study of the Human Spine."

A series of transillumination, consisting of photographs, radiographs and legends descriptive of the anatomy of the bodies of the vertebræ and intervertebral discs and the radiological findings in various diseases involving these structures. An extra trabecular layer of bone reinforcing the lower surface of the vertebral body is noted. The association of this with the usual narrowing of the intervertebral space above a compressed fracture of the vertebral body is desired. An explanation is offered that attempts to reconcile the pathologist's and radiologist's conception of the earliest radiological sign in tuberculous spondylitis, *i.e.*, the narrowing of the intervertebral space.

Dr. A. E. Childe, Montreal Neurological Institute. "Cerebral Pneumography in Childhood."

A series of transparencies, chiefly of radiographs, illustrating various encephalographic and ventriculographic findings in childhood. This will include normal structures, fixed lesions and also expanding lesions. A few other miscellaneous conditions such as congenital abnormalities will be shown.

Metropolitan Life Insurance Co. (Dr. N. L. Burnette), Ottawa. "Pneumonia Treatments and Care."

Twenty Charts showing Etiology, Typing, Serum Therapy and General Mortality Statistics. Exhibit of graphs showing lobar pneumonia—etiology, diagnosis, pathology, specific serum therapy and prognosis; also general mortality statistics for pneumonia.

Drs. G. E. Richards and A. C. Singleton, Toronto General Hospital and Ontario Institute of Radiotherapy. "Exhibit on Radiology."

(a) Diverticulitis of the sigmoid — 60 mounts illustrating characteristic features of the various stages of diverticulitis of the sigmoid. Differential diagnosis and complications. (b) Cholangiography — 60 mounts illustrating visualization of bile ducts by means of lipiodol injection. (c) Myelography — 30 mounts demonstrating normal and abnormals of the subarachnoid space of the spinal canal by means of lipiodol. (d) Non-medical uses of radiography — 25 to 50 mounts, illustrating x-rays of famous paintings including Rembrandt's mother by himself, Egyptian mummies two to ten thousand years old and articles in common use, showing interior structure, etc. (e) Radiological treatment of cancer. A series of mounts illustrating various forms of cancer treated by radiological methods.

Lahey Clinic, Boston.

A series of transilluminated coloured drawings on the following: (a) The two-stage removal of Pulsion Diverticuli of the Œsophagus. (b) Common diseases of the stomach. (c) Subtotal and total Gastrectomy. (d) The Bronchoscopic diagnosis of Carcinoma of the Lung. (e) 1. Trigeminal Neuralgia, diagnosis and management. 2. Subdural Hæmatoma, diagnosis and management. (f) Intra-thoracic Goitre. (g) Sciatica, diagnosis and management.

Dr. F. W. Jackson, Department of Health and Public Welfare, Winnipeg. "Poliomyelitis in Manitoba—1936."

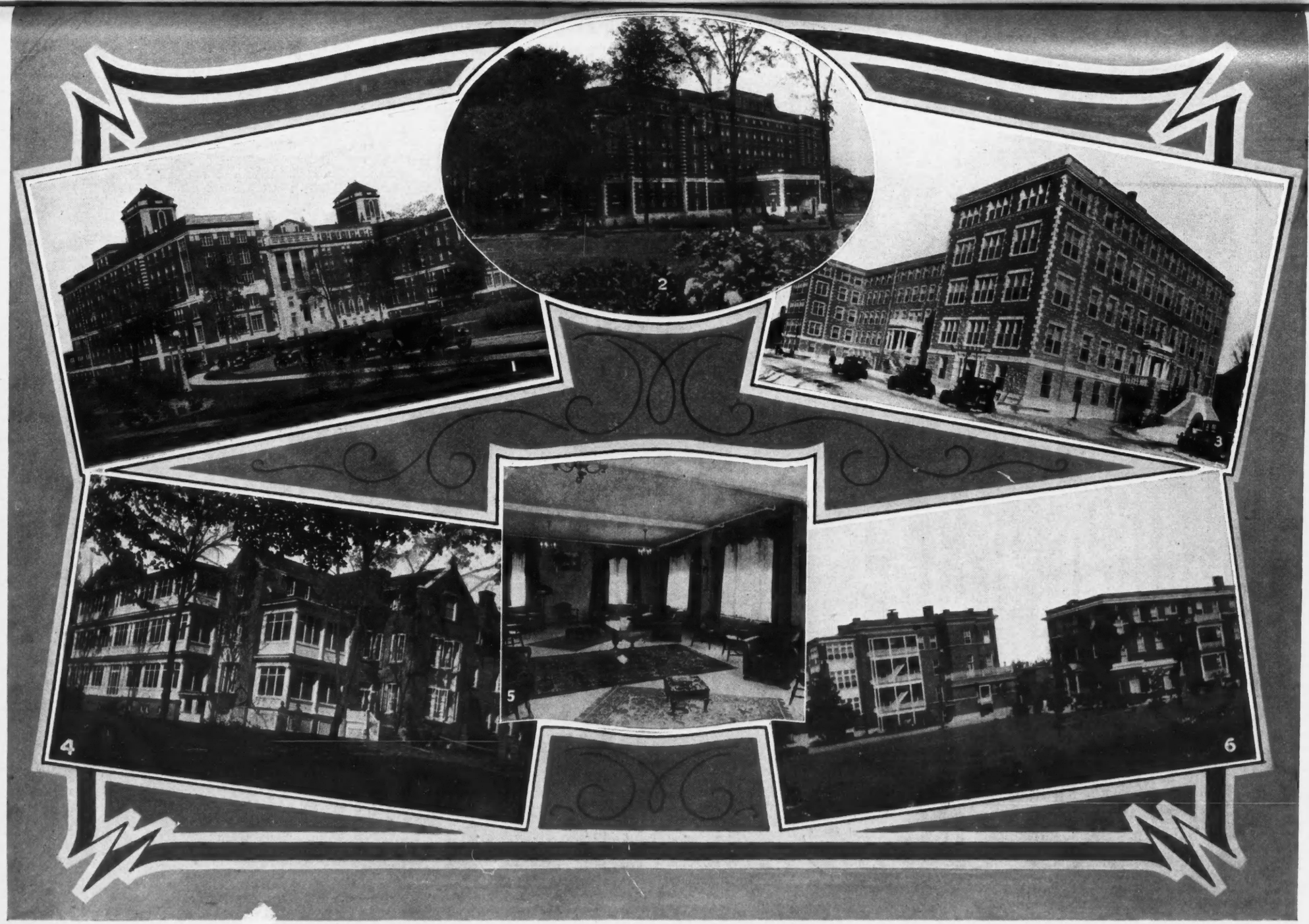
The exhibit of the Manitoba Department of Health and Public Welfare consists of a large map of Manitoba showing the spread and extent of the epidemic of Poliomyelitis in the province in 1936. On each side of the map and connected with central wires to a miniature telegraph pole, it is planned to outline the coordination of effort between the Medical Profession and Municipal and Provincial Authorities in combating the disease.

Dr. L. P. Ereaux, Royal Victoria Hospital, Montreal. (a) "Dermatological Moulages." (b) "Syphilis; Collection of Photographs."

(a) Thirty-six wax moulages illustrating various skin diseases. (b) Forty enlarged photographs illustrating various phases of syphilis.

Dr. H. H. Fellows, Metropolitan Life Insurance Company, New York. "A Program for Control of Pulmonary Tuberculosis in Industry."

Charts and roentgenograms showing the detection of pulmonary tuberculosis among applicants for employment and employees by fluoroscopic and roentgenographic examinations, showing the incidence of disease among the apparently healthy individuals, types of lesions



1. OTTAWA CIVIC HOSPITAL
2. OTTAWA CIVIC HOSPITAL, NURSES' HOME
3. OTTAWA GENERAL HOSPITAL
4. ROYAL OTTAWA SANATORIUM
5. RECEPTION ROOM, NURSES' HOME, OTTAWA CIVIC HOSPITAL
6. STRATHCONA HOSPITAL FOR INFECTIOUS DISEASES

detected and other abnormal conditions found by examination.

Dr. E. A. Watson, Animal Diseases Research Institute, Hull, Que. "Tuberculin; its Preparation and Standardization." "Virus Diseases in Animals."

(a) Cultures of *B. tuberculosis* in synthetic media; separation and ultra filtration of the products and the collection of the pure protein derivative; tuberculin P.P.D.; guinea-pig test reactions. (b) A laboratory demonstration of some virus diseases in animals.

Drs. A. M. Jeffrey and D. A. Carmichael, Royal Ottawa Sanitorium. "Childhood Tuberculosis."

An x-ray demonstration of childhood tuberculosis, consisting of 60 transparencies to illustrate various phases, and the course of tuberculous disease in children.

Canadian Medical Association, (Dr. Harvey Agnew), Toronto. "Canadian Medical Association Exhibit."

Some of the many activities of the Canadian Medical Association will be reviewed.

Dr. R. K. Paterson, Ottawa Civic Hospital. (a) "Exhibit of Skin Malignancies." (b) "Exhibit of Mediastinal Tumours."

Dr. J. A. Hannah, Ontario Department of Health, Department of Neuropathology, University of Toronto. "Pathological Research in Psychiatry."

This exhibit is designed to demonstrate the close relationship existing between psychiatry and other branches of medicine. The material presented consists of various organic findings in autopsy material from psychotic patients. Microscopic sections from whole brains and organs illustrate some of the common organic lesions found. Sudden death in relation to hæmorrhage in the wall of the third ventricle is illustrated. A study of the anatomy and reaction of the dura in relation to subdural hæmatoma is presented. All these conditions are common to psychiatry and the rest of medicine.

Dr. J. W. Gerrie, Montreal General Hospital, Montreal. "Handling Recent Fractures of the Nose."

Recent Nasal Fractures—Drawings, wax and plaster moulages showing types of fracture; demonstration of x-rays of nasal injuries; demonstrations of past and present methods of internal and external fixation or splinting, particularly the internal expanding wire type of splint and the external compound and plaster of Paris splints.

Prof. P. Masson, University of Montreal. "Coloured Micro-photographs on Pathological Subjects."

Miss M. O. Noble. "Canadian Assoication of Occupational Therapy", Toronto.

Describing occupational therapy with children, tuberculous, mental and surgical cases, using photographs, posters, equipment such as bicycle, jig-saw, loom, etc., and some finished work to show how craft work is applied in the treatment of these cases.

Dr. T. R. Little, Ottawa. "An exhibit on Pathology and Bacteriology."

(a) Divided culture plates showing the antiseptic action of the coal tar dye stuffs. (b) Numerous gross pathological specimens.

Dr. F. Risdon, Toronto. "Facial Reconstructive Surgery."

Hospitals in Ottawa

A picture which appears in this number indicates in a general way the facilities which Ottawa enjoys for the housing of the sick. Since the cut was prepared several major additions have been built to the main hospitals included in it. Indeed, since the Medical Convention thirteen years ago hospital expansion has represented a cost of close on six millions of dollars Here, it may not be superfluous to quote from the May number of the *Journal*.

"The several hospitals of the city are well worthy of inspection and will be open for this purpose throughout the week of the Convention. The two leading hospitals, the Ottawa Civic and the Ottawa General, are among the largest and best equipped in the country, and are usually filled to capacity with cases which encourage the academic study of disease. For the convenience of those who might wish to attend routine clinical ward rounds or surgical operations, bulletins indicating the nature of these will be posted daily at the Convention Hotel."

ACCOMMODATION

Now that the date of the Convention is fast approaching, the question of housing grows in importance. It is hoped that the securing of hotel accommodation has not been too long deferred. Consultation with the Chairman of the Housing Committee, Dr. J. H. Alford, 235 O'Connor Street, Ottawa, will be of help in this connection. A constant demand is being made for reservations, but Dr. Alford gives the assurance that comfortable quarters will be found for all. Already he has arranged for the parking (adjacent to the Chateau Laurier), of air-conditioned sleeping cars sufficient in number to take care of any overflow.

Those who intend to use their own cars as a means of conveyance are reminded of the arrangements made for parking in areas, provided for the purpose, through the courtesy of the Federal District Commission.

THE CONVENTION CITY

Much has been said of the forthcoming convention — much of the city in which it is to be held. To attend this convention and to visit this city will be a great privilege to medical and lay people alike. In a peculiar sense Ottawa belongs to all Canadians. At the moment, therefore, it is not inopportune to quote a brief descriptive note which may be read with equal pride by all who live within the borders of our richly-blessed Homeland.

"Rich in tradition and culture, Ottawa, Canada's Capital, possesses an unique charm which sets it apart as one of the world's delightful Capital cities. Its history is closely interwoven with that of the Ottawa River on whose banks it is situated and from which it derives its name. Originally known as the Grand River, it was first explored by Champlain in 1613 when he paused on Ottawa's site to admire its promontories and seething waterfalls. This river, in a few short years, became the fur-trade route to the west and north and, for a century and a half, saw only the picturesque coureurs des bois or swarthy Indians transporting their furs to Montreal.

"It was, however, an energetic, restless American, Philemon Wright, who first visualized the potentialities of the Ottawa District. In 1800 he settled in Hull, opposite Ottawa, and commenced the development of Ottawa's lumbering industry. This was the backbone of its existence for over sixty years. But it was not as a lumbering town that Ottawa became known, rather, as the terminus of the Rideau Canal. The construction of this historic canal was commenced in 1826 by Colonel John By, of the Royal Engineers.

"Wandering in Major's Hill Park, behind the magnificent Chateau Laurier, one may see two stones that mark the site of the Colonel's house from which he directed the building of this canal. Indeed, until 1855, Ottawa was called Bytown after this illustrious Empire Builder.

"Looking from this park at the present time, towards the west, the towering spires of the Parliament Buildings are clearly outlined against the sky. Loud have been the encomiums on the grace, beauty and dignity of the Canadian Houses of Parliament, but to realize how far short all these praises fall one must see them at sunset when the afterglow turns roof and gable, spire and steeple into a vision of beauty.

"It was, however, in 1857 that the then struggling city of Ottawa was chosen by Queen Victoria as the Capital of the Dominion of Canada. Ottawa, meanwhile, has grown and prospered until now it is recognized as one of the world's outstanding capitals. Built on a promontory of the Ottawa River, it commands a fine view of the ever-changing Laurentian hills, and it is a common sight to see native and stranger alike hesitating on the Plaza, just west of the Chateau Laurier, above the Rideau Canal, enthralled by the beauty of this panorama. Almost surrounded by thirty miles of scenic driveway, famed for its shrubbery and particularly for its many varieties of lilacs, Ottawa possesses fine wide streets, bordered by magnificent trees, beautiful homes, and stately buildings, both governmental and industrial. The Parliament Buildings, National Art Gallery, Archives, Bytown Historical Museum, Parliamentary Library, Rideau Hall, the official Vice-Regal residence, Experimental Farm, Dominion Observatory, and many other buildings add greatly to the cultural as well as historic charm of the Canadian Capital.

"As a tourist and convention centre Ottawa is ideal. Reached by motor over a network of excellent highways, it is also the radiating point to a number of vacational districts such as the Rideau Lakes district, Highlands of Ontario, Upper Ottawa River, Gatineau hills and Lievre district, all noted for their fishing and hunting. These districts, within a short distance of Ottawa, are reached over improved highways. Adequate rail, motor coach and air services are available, while the Ottawa and Rideau Rivers, and Rideau Canal furnish excellent yacht and canoe routes from Lake Ontario and the St. Lawrence.

"A city of sunshine, teeming with interest, the Canadian Capital has arrived and proudly takes her place among the leading Capitals of the world."

Hospital Service Department Notes

Federal Government Proposes to Suppress Radio Interference by Electro-therapeutic Apparatus

A memorandum of considerable interest to the medical and hospital world has recently been issued by the Radio Division of the Department of Transport at Ottawa. Numerous complaints have been received by the Radio Division with reference to the operation of electro-therapeutic apparatus. The radiation from short wave therapeutic generators causes much interference in the short wave channels. In Ottawa, for instance, there is so much interference with the Ottawa short wave receiving station as to make it impossible to rebroadcast many desirable programs from Great Britain and foreign countries. Government and commercial communications as well as short wave reception in the home are being interfered with. As tests have shown that radiation from electro-therapeutic apparatus in Canada is carrying to foreign countries, Canada is under obligation, as a member of the International Telecommunication Convention, to protect international radio communications from interference originating in this country. Spark-gap machines have caused most complaints; tube generators radiate in the short wave band and seldom cause interference on the standard broadcast channels. This radiation, however, is intense and affects reception at distances up to several thousands of miles.

The Radio Division is very anxious not to effect a hardship upon the owners of electro-therapeutic apparatus, and, accordingly, is throwing out these suggestions with the hope that ways and means can be developed by manufacturers and users to overcome this interference and thus minimize any difficulties associated with the suppression of radio interference. Very careful consideration has been given to the economic aspect of the situation, and to the value of electro-therapeutic services to the public. Immediate steps to enforce suppression of radiation on frequencies not now

All communications intended for the Department of Hospital Service of the Canadian Medical Association should be addressed to Dr. Harvey Agnew, 184 College Street, Toronto.

required for radio services will not be taken, but protection of such frequency bands will be necessary when they are allotted to radio services.

Authority for action was given by the Canadian Broadcasting Act of 1936, which empowers the Government to enforce the suppression of radio interference (Clause 23). In the interval, the Department has been studying this problem with the cooperation of the Canadian Medical Association, manufacturers, importers and operators of therapeutic apparatus, and now suggests issuing regulations under the Act along the following lines.

TIME LIMITS

January 1, 1938.—No new installations causing or liable to immediately cause interference shall be used after this date. Existing installations may operate without change until January 1, 1939.

January 1, 1939.—Commencing this date, the operation of apparatus causing or liable to immediately cause interference shall be prohibited on Sundays and between twelve noon and twelve midnight, weekdays, and shall be limited to sixty minutes' operation in any twenty-four hours, these conditions to continue for one year until January 1, 1940.

January 1, 1940.—The operation of all apparatus causing or liable to immediately cause interference shall be prohibited after January 1, 1940.

January 1, 1942.—Excessive radiation in the radio spectrum (any frequency used for radio communication) shall be prohibited after January 1, 1942.

Emergency operation.—Emergency operation will not be restricted, but the onus of proof of emergency shall be on the user.

Radiation.—Even with the most effective shielding radiation beyond the confines of the operator's premises cannot be reduced to zero. Such radiation shall, however, be within the intensity limits set by the Department.

Excessive radiation.—This is to be interpreted as radiation beyond the confines of the operator's premises which exceeds the intensity limits set by the Department, including direct radiation and indirect radiation from power wires, etc. Any apparatus causing excessive radiation beyond the operator's premises, on frequencies in the radio spectrum, is liable to cause interference.

Apparatus causing or liable to immediately cause interference. — This class includes: — (1) apparatus actually causing interference, and (2) apparatus causing excessive radiation on frequencies in which there is a licensed radio service within the interference zone. The limits of such zones shall be fixed by the Department.

The Department invites comment on the suggested regulations from all parties interested, and will be glad to give consideration to any changes or amendments that appear desirable.

A CORONATION GIFT.—The employees of A. Wander Ltd., Peterborough, Ont., manufacturers of Ovaltine, were pleasantly surprised when they received a gratuity of one week's salary in commemoration of the Coronation of Their Most Gracious Majesties, King George VI and Queen Elizabeth. Word as to the form being taken in England by the company's recognition of this historical event came from Sir Harry Hague, Managing Director of the Company, in London.

Medical Societies

The Ontario Neuro-Psychiatric Association

The Annual Meeting of the Ontario Neuro-Psychiatric Association was held at the Ontario Hospital, London, on March 18, 1937, with an attendance of 241. The President, Dr. G. H. Stevenson, Superintendent of the Ontario Hospital, London, presided. The meeting was opened with an Invocation by the Rev. K. E. Taylor, of London, and welcome was extended by Alderman E. Curtis, representing his Worship Mayor T. F. Kingsmill, of London. The President then called for a minute's silence to the memory and honour of the passing of Dr. Archibald Kilgour and Dr. Edward Kinsman. Three scientific and interesting papers followed. Dr. J. R. McGeoch, Senior Physician to the Ontario Hospital, London, gave an interesting clinical presentation on "Bromide intoxication". His paper was discussed by Dr. F. J. H. Campbell, Dean of the Medical School, University of Western Ontario, and by Dr. W. D. S. Cross, of the Homewood Sanitarium, Guelph, Ontario. Prof. F. R. Miller, Professor of Physiology of the University of Western Ontario, delivered a clear, scientific and illustrated talk on "Electrical potential changes in the cerebral cortex and other parts of the central nervous system as studied with the cathode ray oscillograph". Dr. D. J. Solandt, Assistant Professor of Physiology in the University of Toronto, discussed his paper. Dr. Milton H. Erickson, Director of Psychiatric Research, Eloise Hospital, Eloise, Michigan, explained and demonstrated scientifically "The applications of hypnosis in psychiatry". His particularly interesting presentation was discussed by Dr. R. B. Liddy, Professor of Psychology, University of Western Ontario, and by Dr. W. J. McLean, Superintendent of the Westminster Hospital, London, Ont.

The evening session was a tribute to the late Dr. Richard Maurice Bucke on the centenary of his birth. Two hundred sat down to the Association dinner. The Chairman called on the guests of honour, members of Dr. Bucke's family, his friends, and his former staff to rise as their names were called. The evening session was held in the auditorium, and Dr. G. H. Stevenson delivered a most fitting, interesting, and appreciative commentary on "The life and work of the late Dr. Richard Maurice Bucke".

The following officers were elected for the coming year: *Honorary President,* The Hon. Dr. J. A. Faulkner; *President,* Dr. J. P. S. Cathcart; *Vice-President,* Dr. D. R. Fletcher; *Secretary,* Dr. A. McCausland. *Executive Committee*—Drs. B. T. McGhie (Chairman), G. H. Stevenson, W. W. Barraclough, J. S. Stewart,

C. S. Tennant, S. G. Chalk, A. L. MacKinnon.
Editorial Committee—Drs. E. A. Clark (Chairman), C. B. Farrar, J. A. Hannah, C. H. Pratt, C. A. Wicks.

The Regina General Hospital

The weekly clinical pathological conference of the staff of the Regina General Hospital discussed a case of typhoid presented by Dr. D. C. McRae in a boy three and a half years old. At first the child was croupy, feverish, delirious, with a temperature of 104° F.; the abdomen was distended. The first diagnosis was pneumonia. White blood cells numbered 3,850. The blood Widal was positive and blood culture negative, the first week. On the second day after admission, rose spots appeared on the abdomen. The child took fluids well at first by mouth, later by nasal drip. A bowel hæmorrhage occurred the third week. Œdema was marked two days before death. Blood culture was positive the third week.

Dr. J. C. Paterson reported the autopsy findings. The blood culture was positive after death. The spleen weighed 150 g. and was bright red. Two Peyer's patches were found in the small bowel. *B. typhosus* was cultured from the gall-bladder and from the base of the two ulcers. There was no focal necrosis of the liver. In the discussion it was stated that 25 per cent of cases have a positive blood culture in the third week of the disease. The mortality is higher, the longer the blood culture is positive. The source of the infection is a woman about 65 years of age who had infected nearly everyone who lives in the same house. This is the second person she has infected this winter.

The second case, presented by Dr. E. A. McCusker, was a 64-year old male who died of streptococcus hæmolyticus septicæmia. He was referred by Dr. D. S. Johnstone for treatment of a nasal polyp. Dr. McCusker removed a piece of the nasal polyp in the office to have it examined for malignancy. Since no malignancy was found, the patient was admitted to the hospital and the polyp removed. The base of the polyp was on the posterior ethmoid quite far back. Dr. McCusker left about a quarter of an inch of polyp. A temperature of 100° F. appeared after the operation, but the man went home on the second day feeling well. Five days after the operation he went to the doctor's office and stated he felt well. Two days later he collapsed. He was clammy and freely perspiring. The following day he showed some rigidity of the neck. Lumbar puncture showed a cell count of 6. The Pandy test was negative; culture was negative. Blood pressure was 190/130. Blood culture was positive for *S. hæmolyticus*. The urine contained albumin and casts. The blood urea was 28.6 mg. per cent.

Dr. Paterson gave the autopsy findings, which were slight chronic nephritis; slight hypertrophy of heart; coronary sclerosis, with almost complete stenosis of the left descending branch; sphenoidal sinusitis; aneurysm of the abdominal aorta, with an abscess in the wall which appeared to be secondary to this infection. In the ensuing discussion the question which remained unanswered was how could sinusitis flare up and cause septicæmia?

Dr. J. Farr then reported 3 cases treated by prontosil. The first case was a *S. hæmolyticus* infection following arthrodesis of the knee. The patient recovered. The second was a case of embolism after appendicectomy, resulting in fluid in the chest from which streptococcus was cultured. This man later developed a bronchial fistula and died. The third was a primipara who after an easy, uncomplicated, three-hour labour developed a temperature of 105° F. on the fourth day. After two days treatment with prontosil, the temperature became normal. She developed cyanosis due to sulph-hæmoglobinæmia. Treatment was stopped for two days, then the temperature again went up to 105° F. The red blood cells numbered 3,000,000. After two transfusions, five days later they numbered 2,000,000. Prontosil was started again. She recovered without pelvic abscess. The blood sulphur was 86 mg. per cent.

Dr. S. E. Moore presented a case of actinomycosis of the appendix in a male, aged 28, a farm labourer. After appendicectomy the wound did not heal. Pus drained for four months. After death, abscesses were found throughout the abdomen. The bodies of two vertebræ were eroded but the intravertebral disc was not invaded. This ruled out tuberculous infection. Dr. Paterson, in discussing this case, stated that he had seen two cases of actinomycosis in Toronto in two years but had seen two cases in Regina in a month.

LILLIAN A. CHASE

Post-Graduate Courses

Opportunities for Advanced Study Open to Foreign Physicians in Berlin

The Berlin Academy for Advanced Medical Study is the centre through which the City of Berlin offers all its opportunities to medical men to pursue their professional studies. Its headquarters are in the Kaiserin Friedrichhaus, Berlin, N.W. 7, Robert Koch Platz 7.

For the foreign doctor Berlin possesses admirable facilities for advanced study, as the Academy is closely allied to all State and

Municipal Hospitals, as well as the private clinics, and to the prominent doctors connected with these institutions. Emphasis should be laid on the great abundance of pathological and anatomical material available for study.

I. International Courses.

In March and October of each year the "International Advanced Courses" are held, including in their staff of instructors such well-known specialists as von Bergmann, Roessle, Sauerbruch, Stoeckel, and Umber. Schedule outlines of these courses may be obtained gratis at the office of the Berlin Academy, Berlin, N.W. 7, Robert Koch Platz 7 (Kaiserin Friedrich-haus). They are given in the German language, and cover the subjects: internal medicine, surgery, obstetrics and gynæcology, tuberculosis, diseases of the throat, nose, and ear, urology and some others.

II. Special Courses in English and French.

For visiting physicians who have not a sufficient command of German, special courses of lectures will be given in English, French, and Spanish. These lectures are in the nature of private courses which will be arranged and prepared primarily with reference to the wishes of the student. There is no set schedule of these courses, but a prospective student has only to acquaint the Academy with the subjects he desires to study, and it will make all necessary arrangements in advance, so that as soon as he arrives in Berlin he may begin his course without losing any time.

These courses can be arranged to last two, four, six, or eight weeks, according to the wishes of the student, and the time he has at his disposal. The fees depend somewhat on the subject-matter, but on an average will amount to 10 marks per hour.

III. Laboratory Accommodation.

The Academy is also prepared to provide laboratory space for those physicians who desire to work in one of the various institutes or clinics for a prolonged period of time, or engage in other scientific work or writing. Such a student is recommended to avail himself of some sort of a work-room or laboratory, as it will give him the advantage of being in closer touch with the clinic, and will enable him to witness operations, assist in observing particularly interesting cases, and study and analyze the result of examinations for himself. Subject to certain formalities, he will also be permitted to work in the chemical, physical, and pathological laboratories, and in this way learn something of modern methods of analysis. Naturally, work of this nature requires a certain amount of familiarity with the German language, as the student must be able to converse with physician and patient alike, but he will always find in the clinics, colleagues who speak his language and who can act as interpreters for him, at least for a time.

In a number of the municipal hospitals, the opportunity is offered to foreign physicians of residing in the hospital for a moderate charge of 2.50 marks per day, including meals. The cost of renting a work-room or laboratory such as above referred to is approximately 100 marks per month, to which must be added the charges for the use of retorts, test-tubes, etc.

IV. Letters of Recommendation and Introduction.

Foreign doctors are advised to call at the offices of the Berlin Academy for Advanced Medical Study upon their arrival in the city, in order to obtain the necessary letters of recommendation and introduction. The Academy office will also gladly assist doctors in procuring suitable lodgings or apartments.

V. The Cost of Living.

This depends almost entirely on what is required. A simple room can be had in Berlin for about 40 marks per month; pension or boarding-house rates range from 120 to 150 marks per month, and a room at a hotel from 3 to 4 marks per day. With 200 marks per month, one can live very comfortably.

VI. Diplomas.

Those attending courses under the jurisdiction of the Academy are entitled to a diploma if they have devoted not less than three months to advanced medical study. For a shorter period of study appropriate certificates are issued.

VII. Other Facilities.

A special convenience is offered to foreign visitors to Germany in the shape of the "registered marks", sometimes called "blocked marks". Drafts in registered marks can be bought at foreign banks, and a visitor is advised to get in touch with his bank or banker *before* starting on his journey.

Attention is also called to the reduction of 60 per cent in railway fares granted to visitors travelling in or through Germany. These reduced rate tickets can be obtained at the foreign offices of the "M.E.R.", (Mittel-Europäisches Reisebuero), and aboard German steamships. All students who have announced their intention to take courses at the Academy will be sent a certificate entitling them to obtain a German visa either free of charge or for a reduced fee.

VIII. Courses in German—Social Introductions.

Brief mention should be made of the "Deutsches Institut fuer Auslaender (German Institute for Foreigners), which is a department of the University of Berlin and makes a

specialty of instruction in the German language and in the outstanding points of interest in Germany's geography and culture.

In the Kaiserin Friedrichhaus is located the "Auslandsamt der Dozentenschaft" (Foreign Department of Teachers) whose function it is to introduce foreign students into German families, where they may have the opportunity of gaining a more intimate knowledge of political and social conditions in Germany. This Department also arranges personally conducted visits to the most interesting and characteristic of the German institutions, to which students at the Academy may be invited upon request.

IX. DOCTORS' INFORMATION BUREAU.

This is situated in the Kaiserin Friedrichhaus, and information on all questions of interest to medical men is freely and impartially given without charge. Every foreign doctor who is planning to study in Berlin would do well to consult this Bureau in advance before arranging his contemplated course of study, so that the same may be arranged in the most convenient and economical manner. This Bureau also gives letters of introduction and recommendation to clinics, institutes, etc.

Topics of Current Interest

The Peking Man

At the close of 1927 news came from China of the discovery of three more skulls of Peking Man (Sinanthropus). In this way Professor Weidenreich, who took charge of the Cenozoic Laboratory in Peking after the death of Professor Davidson Black, came to have six skulls of this extinct race of humanity at his disposal for study—besides fragments—mostly of jaws and skulls—of other twenty individuals. In a recent communication to the *Times* (February 18th) Professor Weidenreich sums up the chief conclusions he has reached concerning the place which must be given to Sinanthropus in the gallery of extinct humanity. Hitherto the pride of lowest place has been assigned to the Java Man (Pithecanthropus). Professor Weidenreich shows cause why China must now oust Java as the home of the lowest known form of humanity. He finds that in brain size a member of the Sinanthropic family had even a smaller brain than Pithecanthropus. Still, this claim will be seen to be not a strong one when it is remembered that among the Australian aborigines of today individuals do occur with a brain as small as that of the smallest Sinanthropus—850 c.cm. Professor Weidenreich's claim of a low position for Peking Man in the scale of humanity does not rest only on small-brainedness. He cites characters which ally this ancient form of humanity—at least half a million years old, having lived at a very early phase of the Pleistocene Period — with anthropoid apes on the one hand and Neanderthal Man of Europe on the other, Sinanthropus being a sort of stepping-stone from the one to the other. Very remarkable, too, is the fact that the Peking men and women differed from each other in size and strength almost as much as a male gorilla differs from the female gorilla. Noteworthy, too, is the range of variation in jaw, tooth, and brain size which prevailed in the humanity of Pleistocene China. In his article in the *Times* Professor Weidenreich puts forward the idea that Mongolian features can be recognized in the anatomical make-up of Sinanthropus and that Peking Man may prove to be the modern Chinaman in the rough. This revolutionary conception will not surprise those who are familiar with the address given by Sir Arthur Keith to the newly founded Speleological Association in the summer of last year. In that address it was maintained that the evidence provided by fossil remains indicates that the chief races of modern man—white, brown, yellow, and black —were already separated early in the Pleistocene, and that since their separation all have undergone parallel changes which have given them their "modern" characteristics—reduction in jaw and tooth, with planing down of their rugosities of skull and limb-bones.—*Brit. M. J.*, 1937, **1**: 455.

Physiotherapy as a Career

Lord Horder, in his address to the students at the opening of the autumn session of Westminster Hospital Medical School ("The Doctor as Humanist", September 28, 1936), gave his listeners much sage advice. He stressed particularly the careers which medical education and qualification opened to those who felt that they were not suited to the actual practice of general medicine, *e.g.*, bio-chemistry, pathology, and biophysics, and continuing with this line of thought, Lord Horder said:

"But the craft may attract you, and, if so, there is great diversity of choice, as there is great diversity of gifts. In the main this may be said to be exploitation of technique through the hand, rather than through the eye and the ear; surgery, to wit: general surgery or special surgery. But the branchings off are by no means exhausted yet. There is radiology,—irradiation in diagnosis and irradiation in treatment,—and already these two are being segregated as separate fields, with, again, irradiation by x-rays as distinct from irradiation by radium.

"Then there is physiotherapy (as distinct from irradiation), the renaissance of which, as an important part of therapeutics, we are now

witnessing,—a welcome and successful salvaging of the various methods concerned from the dark and devious toils of the quack. This rescue of physical methods from the limbo of pseudo-science, and their establishment on sound physiological principles, has opened up a large field for investigation and for exploitation to the advantage of practitioner and patient alike.

"Some specialism is inevitable here also, since the various methods concerned require concentrated study, experience, and technique. Manipulation and massage, actinotherapy, electrotherapy, balneology, scientifically pursued, will sooner or later oust the osteopath, the bone-setter, and the mere turner on and off of electrical and other switches, always provided the indication given by the patient's disease is not in the direction of sheer humbug. In the latter case there is still material for the quack, for, as is said of nations and their government, so patients, for the most part, get the doctors they deserve."

Medico-Legal

XXII.

Dame Rajotte v. X.*

Quebec—Abdominal operation—Compress left in abdominal cavity — Fault — Prescription of action against surgeon.

The defendant had performed an abdominal operation upon the plaintiff in the autumn of 1932. Since that time she had suffered increasing pain, until in the spring of 1934 a second operation was performed by a second surgeon, when a compress was discovered in the abdominal cavity.

Mr. Justice Denis, who presided, held that there could be no doubt that the surgical compress found at the second operation had been left in the abdomen at the time of the first operation, and indeed the defendant had made no attempt to deny the fact. Cases such as these are decided in Quebec under article 1053, which provides that, "Every person capable of discerning right from wrong is responsible for the damage caused by his fault to another, whether by positive act, imprudence, neglect or want of skill". The surgeon, like any other individual, comes within the purview of this article. The question therefore arises whether the omission to remove a compress from a wound amounts to a fault leading to responsibility. On this question the court held that where the conditions of an operation are normal, as here, it is a fault to leave behind a compress that was meant to be removed. There is no fault, or an attenuated fault, only where the operation is performed in circumstances of exceptional difficulty, as for instance in circumstances of urgency complicated by unexpected features such as hæmorrhage. No proof having been offered of any exceptional circumstances, the court awarded the plaintiff $3,000 with interest and costs. On the purely legal question of prescription it was held that article 2262 of the Quebec Code, setting up a prescriptive period of one year, had no application to the present case.

* (1936) Q.R. 74 S.C. 569.

XXIII.

Children's Memorial Hospital v. Davidson.*

Quebec—Claim by hospital for services rendered to infant son of defendant.

The plaintiff by the present action claimed the costs of hospitalization and of an operation performed upon the defendant's minor son. The defence was that the child had been taken to the plaintiff hospital against the father's wishes and without his authorization, that he had had no dealings with the hospital in the matter, and that he owed nothing. From the evidence it appeared that the boy had been born with a malformed palate in a hospital in Noranda, and with the financial assistance of the Kiwanis Club of Noranda had been moved to Montreal for operation. The defendant had not been consulted about this. The only part he took in the whole affair was when, at the request of the Kiwanis, he signed a consent authorizing the operation. His allegation that his signature had been obtained upon the assurance that he would have nothing to pay had not, however, been denied.

It was held, first, that the services had never been requested by the defendant as alleged by the plaintiff, and that in consequence no contract existed between them. Secondly, whatever might have been the situation had the operation been essential for the saving of life, this particular operation was only designed to correct a defective palate, and actually had been of no benefit. One cannot by the intervention of a third party obligate a man to pay for something from which he had not benefited. The services claimed for were, in short, neither necessary nor beneficial. The action was in consequence dismissed. (G.V.V.N.)

* (1935) Q.R. 74 S.C. 268.

Abstracts from Current Literature

Medicine

Erythema Nodosum. Holmes, A. H., *The Practitioner,* 1937, **138**: 181.

The author reviews the facts relating to the etiology of erythema nodosum as recorded by various observers. A careful study of the literature showed the influence of season to be slight and that the disease occurred in all age periods. Three theories of etiology are discussed; the first, and untenable, that it is an acute and specific disease, classified with the exanthems; secondly, that it is a manifestation of tuberculous infection; and, thirdly, that it is associated with rheumatic infection. The evidence of other writers amounts to proof that in children both streptococcal and tuberculous infections may produce erythema nodosum, tuberculosis being the more frequent factor. The author reports a series of 40 cases in adults, of which, for reasons as yet unexplained, 38 were in women, and concludes that in adults the majority of cases are due to rheumatism. Twenty-six of the cases, or 65 per cent, gave evidence of more than one of the generally accepted manifestations of rheumatic infection, either in their previous history or concurrently with erythema nodosum. Treatment of the condition should be directed toward the relief of pain; a protective cradle, local applications such as lead and opium mixtures, appropriate sedatives, and salicylates being recommended.

DAVID RODGER

Intake of Potassium, an Important Consideration in Addison's Disease. Wilder, R. M., Kendall, E. C., Snell, A. M., Kepler, E. J., Rynearson, E. H., and Adams, M., *Arch. Int. Med.,* 1937, **59**: 367.

Loss of sodium ions often is the cause of serious crises in Addison's disease. The addition of sodium citrate to the treatment has helped considerably. A further step in the control of electrolytes in the blood was to reduce to a very low level the potassium in the diet, and this apparently made it possible to manage the disease without extracts of adrenal cortex. Conversely, adding even small quantities of potassium to the diet had a very bad effect.

Three patients with Addison's disease were used for this investigation. All three did very well with a low potassium diet and sufficient sodium ions, even when extracts from adrenal cortex were withheld. Periods of successful treatment were possible without lowering the potassium intake so long as a generous allowance of sodium chloride and sodium citrate was given. The theory is advanced that normally potassium ions are controlled by the adrenals. Some writers attribute the asthenia of Addison's disease to the toxicity of the potassium ion, and go on to suggest that this can be applied in the diagnosis of Addison's disease where the nausea and wasting means drawing on the reserve of tissue protein which is high in potassium.

P. M. MACDONNELL

Active Immunization Against Tetanus. Brown, H. H., *Brit. M. J.,* 1937, **1**: 494.

Ramon and Zoeller immunized several hundred human beings with tetanus toxoid, and subsequently found an appreciable antitoxic serum content, which had not completely disappeared in four years. Sneath and Kerslake examined the sera of 13 persons inoculated subcutaneously two years previously with tetanus toxoid and demonstrated specific antitoxin. Another dose of toxoid at this point produced a marked rise in a short period of time. Glenny found that the addition of alum to tetanus toxoid doubled the value of the antitoxin produced in horses, and Bergey also noticed this in guinea pigs. A French Army Commission strongly advocates wholesale immunization of troops. The conclusion seems to be that the injection of toxoid, or preferably alum-toxoid, confers a complete and permanent immunity in the majority of those tested. The author recommends for the British Army two doses of alum-toxoid at three months' interval, with a third inoculation before foreign service or in the event of war. A combination with the routine T.A.B. vaccination would be advantageous, according to work by Ramon and Zoeller.

ARNOLD BRANCH

Surgery

Cruciate Ligament Injury. Cubbins, W. R., Callahan, J. J. and Scuderi, C. S., *Surg., Gyn. & Obst.,* 1937, **64**: 218.

This paper deals with 16 cases of rupture of the cruciate ligaments, single or double, associated with partial or complete dislocation of the knee-joint.

Diagnosis of cruciate ligament injury is best elicited under general anæsthesia at the time of injury. Loss of stability and marked passive mobility in the anterior-posterior direction, with lateral or medial deviation of the extended leg, are to be considered pathognomonic. Nine of these cases were seen early. The authors had perfect results with several cases sutured with catgut or silk, immobilized by plaster of Paris cast in 25 degrees flexion for one month, and straight line extension for 3 months. One patient danced at the eighth week without injury. Other cases immobilized for a similar period without cruciate ligament suture did just as well.

In the cases seen late with persistence of signs of cruciate ligament instability the authors found healing of the tibial and fibular collateral ligaments and no sign of the presence of the cruciate ligaments. They believe this to be due to lack of sufficient immobilization. They give the details of the anterior approach through the vastus intermedius and the lateral approach through the vastus lateralis for replacement of the cruciate ligaments by fascial strips. By means of periosteal-synovial flaps on the femur and tibia and drill-holes in the condyles of the femur these fascial strips are placed in positions which were approximately those of the cruciate ligaments. Immobilization was carried out as in the early operative cases. The authors expect complete functional use in one year with the use of a brace or support after the 6th or 7th month. FRANK DORRANCE

The Formation and Treatment of Calculi in the Biliary Ducts and Gall-bladder. Walton, Sir J., *Surg., Gyn. & Obst.*, 1937, **64**: 257.

This address was given as the annual Oration on Surgery before the Clinical Congress of the American College of Surgeons.

The author hypothecates a chronic inflammation of the gall-bladder wall as essential to the formation of pure cholesterol and mixed stones; in the former case the bile does not flow freely through a partially occluded cystic duct as it does with the latter. He does not support the contention of pure cholesterol stones being formed as a deposit from a supersaturated solution; he has never seen a pure cholesterol stone in the markedly distended gall-bladders secondary to common duct obstruction associated with carcinoma or chronic pancreatitis. The presence of pigment stones (biliverdin and bilirubin) is indicative of metabolic disturbances, and they will frequently recur after removal unless thorough medical care is given. And, in this regard he believes olive oil is worthy of trial. He has not any explanation to offer for the formation of calcium carbonate stones.

Sir James finds *B. coli* as the probable source of infection with pregnant women. Streptococci, pneumococci, *Cl. welchii, B. typhosus* and the common influenza bacillus are the other frequent infecting microorganisms. These infections are usually blood-borne, but may be carried to the gall-bladder in the lymphatic system.

He reminds us of the devious channels through which oral dye must pass to give a positive x-ray shadow. Any interference with absorption by the intestine, excretion by the liver, passage through the hepatic and cystic ducts, or concentration in the gall-bladder, may result in a negative shadow.

He does not consider the presence of pigment stones or mild post-prandial gaseous eructations sufficient indication for operative measures. They are indications for a medical regimen. In young healthy persons, in associated angina pectoris or other cardiac lesions, in increasingly frequent colic attacks (even in poor risks), in major discomforts and imminent complications, operation is indicated. He would treat all acute cholecystitis cases associated with gall-stones by early cholecystectomy, with exploration of the common duct and dilatation of the ampulla and sphincter, except in stout unhealthy persons in whom no stone can be palpated in the common duct and whose cystic ducts are of small calibre. He is particular to define all bile-ducts before releasing adhesions. He uses a drainage tube down to the stump of the cystic duct and believes it to be a good safeguard in case a small accessory bile-duct is not recognized. He uses the same procedure as in his acute cholecystitis cases for impacted stone in the common bile duct, and finds he has less often to insert a plain tube, sutured with plain catgut, into the common duct for the usual 10 days. He finds the T-tube is difficult and dangerous to withdraw.

FRANK DORRANCE

Obstetrics and Gynæcology

Variations of Weight During Pregnancy. Evans, M. D. A., *Brit. M. J.*, 1937, **1**: 757.

The average increase in weight during pregnancy is 16 to 18 pounds, and the monthly gain during the second and third trimesters is normally between 2 and 4 pounds. An increase of 5 pounds or more per month is suggestive of an impending toxæmia, the probability of its occurrence varying directly with the amount of the sudden increase. Every patient should weigh herself regularly throughout pregnancy, and be instructed to consult her doctor if the monthly gain is 5 pounds or more. Systematic weighing of patients during the ante-natal period is of definite value in foretelling, and perhaps avoiding, the toxæmias of pregnancy.

ROSS MITCHELL

Placental Variation. Calkins, L. A., *Am. J. Obst. & Gyn.*, 1937, **33**: 280.

A study of some 2,000 consecutive deliveries for the significance of placental weight and placental area in their relationship to maternal height, weight and age, the duration of pregnancy, the parity, the fetal height and weight, and the loss of blood in the third stage of labour indicates that there is no relationship between these measures of placental size and those of the other factors, except in regard to fetal height and weight and duration of the pregnancy. Possible relationships between placental size and maternal weight are so small

as not to possess any considerable clinical significance.

ROSS MITCHELL

Progesterone in Pre-eclamptic Toxæmia. Robson, J. M. and Patterson, S. J., *Brit. M. J.*, 1937, **1**: 311.

Certain experiments in which toxic conditions leading to death were produced in pregnant rabbits are described. These conditions were associated with a failure in the secretion of the luteal hormone. The results following the administration of progesterone in 12 patients suffering from severe pre-eclamptic symptoms are given. Progesterone was given in 5 mg. doses daily for the first three or four days, and, thereafter, depending on the response, at longer intervals. It is evident that the numbers are too small to establish formal proof of the efficacy of the treatment, and the results are published at this stage because it would take many years to collect adequate data in a single hospital. The results appear to be sufficiently encouraging to deserve testing on a wider scale without delay.

ROSS MITCHELL

The Early Diagnosis of Abruptio Placentæ and Its Treatment with Wheat Germ Oil. Shute, E., *Am. J. Obst. & Gyn.*, 1937, **33**: 429.

Many cases of abruptio placentæ are overlooked because the early symptoms are not recognized. Prominent among these are localized uterine tenderness and steady sacral backache. The placentas from such cases are usually small for the duration of the gestation, and almost always display one or several marginal lesions, ranging in character from fresh blood clot to recently organized leached clots resembling cheese in consistency, or even appearing as white infarcts. Seventy-eight per cent of 82 cases of mild and severe abruptio placentæ showed serological evidences of deficiency of vitamin E. Wheat germ oil was used prophylactically and therapeutically with satisfactory results.

ROSS MITCHELL

Urology

The Influence of Infection of the Lower Urinary Tract and Reproductive Organs on the Kidneys: with Special Reference to Lithiasis and Hydronephrosis. Winsbury-White, H. P., *J. Urol.*, 1936, **36**: 469.

The author has attempted in a dramatic experimental manner to correlate clinical pathological findings in the upper urinary tract with infective lesions of the lower urinary and genital tracts, and to obtain information as to the routes along which infection from the genital and lower urinary tracts travel to the kidneys. India ink, living and dead tubercle bacilli were, in different experimental animals, injected into the periurethral tissue, base of the bladder and uterine cervix. Serial sections were then made of these tracts and studied microscopically. Certain phenomena were characteristic of all experiments, namely, the foreign material became collected outside the blood vessels supplying the injected area, apparently in lymphatic pathways to the regional glands; the ureters failed to demonstrate any contiguity of lymphatic channels but drained to regional glands at different levels. However, with one exception (injection of ink into the cervix), on approaching the ureteropelvic junction, the ureter showed a marked perivascular round cell infiltration and collection of the foreign dye material. The same findings were noted in the renal pelvis. Peculiar to the experiment in which injection of the ink was made directly into the bladder wall, it was possible to demonstrate a contiguity of the dye up the lymphatic channels of the posterior abdominal wall directly into the kidney. In the instances where the dye was injected into the servix and periurethral tissues there was a dissemination of the dye particles into the deeper structures of the bladder floor with a desquamation of the overlying epithelial lining. There was no sign of the dye reaching the kidney, but the conjecture is added that, once in the bladder structures, there is obvious reason that it should eventually follow the previously mentioned course. This was suggested by the perivascular collections of inflammatory cells found in the kidney as a result of injections into the cervical tissues of tubercle bacilli.

The author is of the opinion that symptoms of urinary tract disturbances, characterized by frequency of urination, dysuria and loin pain, showing evidence of mild degrees of hydroureteronephrosis can be ascribed to lower genital tract infections.

J. V. BERRY

Hæmorrhagic Cyst of the Kidney. Barney, J. D., *J. Urol.*, 1936, **36**: 602.

Hæmorrhagic cyst of the kidney is an outstanding condition not only because of its rarity but because of its obscure and varied symptomatology. Many theories are offered as to the etiology. Some authors believe these cysts to be bizarre forms of hæmatoma or a type of aneurysm. Others consider that they arise from embolic or thrombotic infarction. Trauma has also been considered and hæmorrhage into a simple cyst of unknown origin has been considered an important factor.

There may be no symptoms, subjective or objective, previous to the onset of the hæmorrhage. In one case-report the symptoms at the

time of onset were suggestive of renal colic, as well as of a rather severe but obscure hæmorrhage. The character and location of the pain in this case and the tenderness evidenced was not characteristic of the usual findings in renal disease. In contrast to this, the author's second case had no signs or symptoms during life which could be elicited by history or physical examination. At autopsy a small hæmorrhagic cyst with a markedly calcified wall was found. The factors which bring about their recognition are pressure, pain, the presence of a tumour, intracystic pressure or acute hæmorrhage. Pyelography will assist in the diagnosis.

J. V. BERRY

Ophthalmology

Ocular Symptoms of Suprasellar Meningiomas. Hartmann E. and Guillaumat, L., *Ann. d'Ocul.*, 1937, **174**: 1.

In their development suprasellar meningiomas press on the optic nerve and the chiasma, and by so doing cause visual disturbance, which is one of the first clinical signs and of the important elements in prognosis. The condition is often confused with retro-bulbar neuritis of toxic or infectious origin. Surgery offers the only possible treatment and gives good results. The vision is improved, and as meningiomas are histologically benign they do not become generalized, while a local recurrence, if it does occur, comes on very slowly. Cushing states: "When the question 'What's wrong with you?' is put to adult patients who are referred to a neurosurgical clinic, the answer 'My sight is failing' is probably in these latter days far more often given than any other". The failure of central vision and the contraction of the peripheral visual field should direct us to the examination of the fundus of the eye immediately.

In this article a summary of the findings in 21 patients is given. The diagnosis was verified microscopically and histologically in all. The article is illustrated with drawings and tables; bibliography.

S. HANFORD MCKEE

Observations on the Clinical Interpretation of the Ocular Tension. Bailliart, P., *Ann. d'Ocul.*, 1936, **173**: 945.

Following a recent communication at the Ophthalmological Society of Paris, where Merigot, of Treigny, justly defended the operative treatment of glaucoma, the question has naturally been raised again as to the most opportune time for operation. Is this when hypertension commences? What is the highest limit of physiological tension that we know of? Morax, an authority whom we always like to quote, wrote in 1921, "We consider as normal, that is, having no harmful consequences, a tension between 15 and 28 mm. Hg. In fact, since I have examined the globes with the Schiotz tonometer I have never met with glaumatous symptoms in eyes having a tension of less than 30, and have never considered as pathological any aggravation in glaumatous eyes up to 28". Does this signify therefore that the physiological ocular tension lies between 18 and 28 mm. Hg?

The author gives in detail his experiments with the tonometer on patients in his clinic, and divides the cases into three groups: (*a*) In the first group the ocular tension remains as at first taken, the needle moving between 25 and 23; (*b*) in the second group the needle continues to move but tends to move downward, 24-22, 23-21, etc.; (*c*) in the third group the reverse is produced; the needle continued to move, but upward, 34-30, 36-32.

Too much importance must not be placed upon the tonometer; although it is an excellent instrument its value is relative. To establish a diagnosis, prognosis and treatment we must not depend upon figures but search for symptoms, and to relieve symptoms the cause must be found.

S. HANFORD MCKEE

Radiology and Physical Therapy

The Treatment of Selected Cases of Chronic Catarrhal Deafness by X-rays. O'Brien, F. W., *Radiology*, 1937, **28**: 1.

A series of 140 cases of chronic catarrhal deafness was treated by a standard x-ray formula:—145 KV., 5 M.A., 0.25 mm. cu. 1 mm. al. filter, 50 cm. distance, 15 x 15 cm. field, 5 minutes, about 90 roentgens in air to each field at each sitting, repeated at weekly intervals for nine treatments. Seventy-three patients were improved. Eighteen out of a group of 20 with tinnitus were cured. The good results are perhaps due to direct effect on lymphoid tissue or due to a reduction in the bacterial content in the throat, for it is known that lymphadenoid tissue is highly radiosensitive, and the response of chronic inflammation to x-radiation now seems well established. Improvement in hearing, when it occurs, is sometimes astonishingly speedy, or may be delayed, appearing after several treatments in progressive steps.

A. STANLEY KIRKLAND

Treatment of Otitis Media and Mastoiditis by the Roentgen Ray. Lucinian, J. H., *Am. J. Roentgenol.*, 1936, **36**: 946.

Fifty consecutive cases of otitis media (31 acute, 8 subacute, and 11 chronic) were treated by roentgen therapy over the ear and mastoid area. The treatment was consistently followed

by relief of pain, increased discharge, improved hearing, and amelioration of systemic manifestations of disease. None of the acute cases developed mastoiditis or perforation of the drum after x-ray treatment, and none of them required tympanotomy. Mastoiditis was already present in 9 cases before x-ray therapy was applied. Two of these later required mastoidectomy. Roentgen therapy properly applied in acute and chronic otitis media shortens the course of disease and prevents complications. No deleterious effects were observed.

The technique advocated is 100 to 110 Kv., 16 inch distance, 2 mm. aluminum, 5 M. A., 5 minutes; tube output, 14.3 roentgens per minute. The total dose is 71.5 roentgens, measured in air; usually a single dose is sufficient; repeated doses may be necessary in subacute and chronic cases.

A. STANLEY KIRKLAND

Therapeutics

The Treatment of Seasickness. Hill, J., *The Practitioner*, 1937, **138**: 297.

The writer classifies seasickness according to the predominating signs and symptoms into three groups; firstly, those cases showing a heightened degree of vagus tone, (vagotonia); secondly, where increased activity of the sympathetic nervous system is manifested, (sympatheticotonia); and, thirdly, a combination of these two groups. With vagotonia, headache, vertigo, and gastro-intestinal "squeamishness" are the prominent symptoms, vomiting being less frequent. The pulse is slow, often less than seventy, and there is usually hypotension. With sympatheticotonia, nausea, vomiting, and retching occur; headache is less frequent, usually frontal. The face is flushed, the pulse usually above 80, and there is a slight degree of hypertension. Treatment should be preventive and active. In preventive treatment, suitable sedation and adequate sleep before commencing the voyage, together with reassurance, especially in the case of the tyro, are of value in improving the mental attitude of the patient. During the voyage an adequate amount of fresh air, warmth, and a reduced diet, which, if anything, should favour the carbohydrates, are advocated. In cases of vagotonia, good results are obtained from the use of a mixture containing Chloral Hydrate, gr. xv; the Triple Bromides, gr. xl; and Tincture of Belladonna, min. xv, suitably flavoured, with water to an ounce, and given in dram sips every half hour when necessary. The amount of the first two drugs may be increased, while decreasing the amount of the latter, for use in cases exhibiting sympatheticotonia.

DAVID RODGER

Para-aminobenzenesulphonamide and its Derivatives: Clinical Observations on their Use in the Treatment of Infections due to Beta-hæmolytic Streptococci. Long, P. H. and Bliss, E. A., *Arch. Surg.*, 1937, **34**: 351.

The preliminary observations of these authors confirmed the report of Colebrook and Verny, that prontosil solution had little if any effect on hæmolytic streptococci *in vitro* but that a concentration of 1:10,000 para-aminobenzenesulphonamide had a marked static action on beta-hæmolytic streptococci when tested *in vitro* in the presence of beef infusion broth or 50 per cent normal horse serum broth. Long and Bliss used prontosil for injection into the tissues and prontyelin tablets for oral administration. These are trade names for para-aminobenzenesulphonamide.

Experimental observations on the mode of action on these compounds lead them to believe that they act by inhibiting the growth of beta-hæmolytic streptococci and that they injure these organisms in such a way as to permit them to be phagocytosed by the white cells.

In a group of 71 patients treated at the Johns Hopkins Hospital with this drug, there were only four deaths. The types of infections treated were erysipelas and cellulitis, septicæmia, puerperal sepsis, traumatic orbital cellulitis, osteomyelitis, cystitis, peritonitis. tonsillitis, peritonsillar infections, scarlet fever, Ludwig's angina, otitis media. The authors state that the clinical results obtained in the treatment of human beings with infections produced by beta-hæmolytic streptococci have been striking.

G. E. LEARMONTH

Intensive Collapse Therapy in Pulmonary Tuberculosis: 1. A Study of the Extent and Results of Such a Program in a Group of 1,124 Patients. Leslie, G. M. and Anderson, R. S., *Am. J. M. Sc.*, 1937, **193**: 149.

The authors have carried out a statistical study of the results of tuberculosis treatment on patients in the Michigan State Sanatorium admitted over a four-year period from June, 1930, to June, 1934. In all 1,124 patients were included in the series of whom 823 were discharged at the time the investigation was conducted. The remainder, 301 in number, were still under treatment in the institution. Collapse therapy was employed in approximately three-quarters of the entire group. Of the 595 discharged patients who received collapse therapy the disease was arrested or apparently arrested in 55.4 per cent; favourable results were obtained in 77.8 per cent; cavity closure in 71.3 per cent of cavity cases; closure or decrease in size of cavities in 84.2 per cent; sputum conversion in 72.7 per cent of the posi-

tive cases; and a negative sputum in 79.3 per cent of all discharged cases.

A comparison of the foregoing results of intensive collapse therapy with those of a large number of sanatoria using relatively little collapse therapy clearly showed that the intensive program was vastly superior from every point of view. These conclusions are further strengthened by the fact that the Michigan State Sanatorium admitted a higher percentage of cases in whom the disease was far advanced at the time of admission.

E. S. MILLS

Pathology and Experimental Medicine

Significance of the Obstructive Factor in the Genesis of Acute Appendicitis: An Experimental Study. Wangensteen, O. H. and Bowers, W. G., *Arch. Surg.*, 1937, **34**: 496.

Information concerning the nature of the origin of appendicitis remains almost as obscure as when Fitz made his prophetic pronouncement. In the last twenty-five years there has been an increase in the mortality rate of 30 per cent. It is obvious that the accomplishment of the surgeon in dealing with acute appendicitis has not kept stride with his potential capacity for coping with the problem. Infection and obstruction are commonly acknowledged as being likely etiological agencies, infection playing the dominant rôle. The specific infection in the alimentary canal is so unusual as to suggest that the anatomical plan of the vermiform appendix may be largely responsible for the frequency with which appendicitis occurs in man. With this consideration as a premise, the authors have tried to evaluate the significance of the obstructive factor in the genesis of appendicitis, by experiments on animals and by careful observation of the frequency with which obstructive phenomena are noted in the spontaneous occurrence of the disease in man. The results of these experiments point towards two important agencies in the occurrence of appendicitis, *viz.*, (1) obstruction, (2) infection. During 1935 the operative specimens of 91 patients with conditions diagnosed pathologically as acute appendicitis were available for examination. There were 43 cases of acute suppurative appendicitis, 19 cases of non-perforative gangrenous appendicitis, and 29 of perforative gangrenous appendicitis. Of the 43 cases of acute suppurative appendicitis an appendiceal concretion had caused a definite obstruction in 18, or 44 per cent. In the 13 additional cases obstruction appeared to have developed through the agency of a stricture, a sharp villus or an abnormal band.

The authors conclude that the essential inciting factor would appear to be a disturbance in the pressure-distension relationship of the appendix. There appears to be a sphincter-like mechanism at the base of the appendix, which makes of it a potential closed loop, with all its attendant inherent dangers. Appendiceal concretions are found as acutely obstructing agents in most instances of perforated appendices.

G. E. LEARMONTH

Parathyroid Hyperplasia in Rabbits Produced by Parenteral Phosphate Administration. Drake, T. G., Albright, F. and Castleman, B., *J. Clin. Investigation*, 1937, **16**: 203.

From the observations of Bergstrand and others the fact has become established that some cases of renal insufficiency are accompanied by enlargement of the parathyroid glands. This was well exemplified by a case recently studied at the Massachusetts General Hospital. The question as to the cause of the parathyroid enlargement arises. Albright, Baird, Cope and Bloomberg have suggested that the determining factor might be the retention of phosphate in chronic renal insufficiency. Binger found that when the level of the blood phosphate is raised by the intravenous administration of phosphate hypocalcæmia and tetany ensue. It seemed not unlikely to the authors that either hyperphosphatæmia or the resulting hypocalcæmia might be the stimulus for the hyperplasia of the parathyroids. The present investigations were undertaken to determine the effect of the parenteral administration of phosphate on the parathyroid glands of rabbits. The authors found that the average weight of the inferior pair of parathyroid glands of 19 control rabbits was 13 mg., while the corresponding figure for 19 rabbits which had received injections parenterally of phosphate three times daily was 20 mg. Histological sections of the latter glands showed definite evidence of hyperplasia. These findings support the hypothesis that retention of phosphate is the cause of the parathyroid hyperplasia in cases of chronic renal insufficiency. Further studies will be required to show whether the hyperphosphatæmia causes the hyperplasia directly or indirectly by producing hypercalcæmia.

JOHN NICHOLLS

Pathology of Pregnancy Toxæmias. Zimmerman, H. M. and Peters, J. P., *J. Clin. Investigation*, 1937, **16**: 397.

The purpose of this study was to determine the cause and nature of the toxæmias of pregnancy, and it is based on the clinical features of 309 cases diagnosed as toxæmias of pregnancy and 11 in which renal or vascular disease had apparently arisen during pregnancy. Autopsy notes are given of 23 cases and the paper is well illustrated. The authors' conclusions are as follows.

Although characteristic tubular and glomerular lesions were usually found in the kidneys of patients who died in the acute stages of toxæmias with eclamptic syndromes these lesions, like hepatic necroses, were not found exclusively in eclampsia. Eclampsia could not be distinguished from other toxæmias on the basis of either pathogenesis or morbid anatomy. "Malignant nephrosclerosis" was frequently found. Both acute nephritis of infectious origin and antecedent vascular disease apparently gave rise to toxæmias which sometimes presented eclamptic manifestations. A great variety of vascular and renal conditions may act as predisposing causes of toxæmias. Pregnancy appears to give them a distinctive coloration and an explosive character. JOHN NICHOLLS

Multiple Polyposis of the Colon: Familial Factor and Malignant Tendency. McKenney, C., *J. Am. M. Ass.*, 1936, **107**: 1871.

The reports of two families with multiple polyposis and cancer of the colon, here recorded by McKenney, should answer the doubts of those who feel that the hereditary factor in cancer is not obvious in the human being. McKenney found two families in which multiple polyposis and cancer of the colon existed in such a large proportion of the members as to preclude any interpretation other than that the disease was hereditary. Family A consisted of 15 persons, 13 of whom were examined. The grandfather died at 47 of bowel trouble, suggestive of cancer of the rectum or colon. Of his 6 children, 4 had died of multiple polyposis and cancer at the ages of 42, 41, 30 and 24. One daughter was normal, and 1 aged 20 had polyps but had not developed cancer. Of the 8 children of the 4 persons who had died, 2 aged 16 and 10 had multiple polyposis. The other 6 were under the age of 11.

The second family consisted of three generations. The grandfather died at 44 of multiple polyposis and cancer of the colon. Of his 7 children 1 died at 34 of bowel trouble suggestive of rectal cancer, and 2 others, aged 29 and 21, had died of multiple polyposis and cancer. The third generation was composed of 20 children, all under 18. Of these 6 had been found to have polyposis. They ranged in age from 11 years to 2. The 4 children of the woman dying of cancer at 29, born to her in two different marriages, all had polyposis. As malignancy apparently always develops in these polyps if the patient lives long enough the outlook is not bright.

In the discussion, it was pointed out that a colostomy on such young patients was not advisable and that x-rays did not always help the condition. One surgeon advocated a temporary cæcostomy until the œdema about the polyps subsided, and fulguration of the polyps at fairly short intervals so as to destroy them before they became malignant, and then, finally, an anastomosis between the ileum and sigmoid, with eventual removal of the colon in the affected area. One such patient was alive and well after four years following such treatment. MADGE THURLOW MACKLIN

Obituaries

Dr. Joseph Alexander Bell, of Sarnia, Ont., died on April 12, 1937, aged 68. He was born in Strathroy, Ont., and a graduate of the University of Western Ontario (1897).

Dr. Bell entered municipal life in 1907, when he was elected councillor for the then town of Sarnia. He was reelected in 1908 and 1909, during these two years serving as chairman of the market and license committee. In 1913 he was elected mayor by acclamation. He served with the C.A.M.C. in France during the Great War.

Dr. Bell received his appointment as medical officer of health and city physician in May, 1925, succeeding the late Dr. William Logie.

Dr. Joseph Moise Brisebois, of Longueuil, Que., died on April 12, 1937. He was in his 77th year, and had practised in Pierreville, Que., and later in Longueuil, since 1883. Born in St. Michel d'Yamaska, he received his education at Nicolet School and at Victoria University (M.D., 1883).

Dr. William Wallace Chipman, of Vancouver, formerly of Ottawa, died on March 29, 1937. He was born at Elgin, Ont. He began practice in the Yukon and served overseas with the C.A.M.C. After the war Dr. Chipman resumed his practice in the Yukon until 1924, when he came to Vancouver.

Dr. James Eldon Craig, of Ottawa, died on April 7, 1937, in his 69th year.

A native of North Gower, Ont., he received his early education in Ottawa, and graduated in medicine from McGill University in 1889. Shortly after his graduation he started to practise in Ottawa, where he continued for more than 35 years. He had been County Coroner since 1905.

Dr. James Fraser Ellis, of Ottawa, died on April 3, 1937.

Dr. Ellis was born at Upper Stewiacke, N.S., on June 11, 1871, the son of William and Margaret Fraser Ellis. He received his primary education in the public school and later at the famous Pictou Academy. Going west after graduation he taught in the public schools for a short period and then entered upon the study of medicine at Western University from which he graduated in 1898. Soon after he returned to Nova Scotia and engaged in practice at Sherbrooke. In 1904 he was elected a member of the House of Assembly. He was reelected in 1906 and 1911. In the next House he was elected Speaker, and continued in that office until he went overseas in 1916. He joined the St. Francis Xavier's Hospital Unit in Antigonish with which he proceeded to France. Later he was transferred to the Dalhousie Hospital Unit and saw extensive service in the war zone. During the period of demobilization he was medical officer at the Bushy Park Hospital in England.

Dr. Ellis returned to Canada in April, 1919. He was then attached to Camp Hill Hospital, Halifax, as District Medical Adviser. In 1923 he was appointed a

member of the Board of Pensions Commissioners of which Colonel John Thompson was chairman. He was reappointed to the reorganized Canadian Pensions Commission in 1935 and was the senior member of the Board, to which he gave for a period of 14 years devotion combined with unfailing sympathy and good judgment in his administrative duties.

Dr. Henry Grundy, of North Toronto, died on April 8, 1937. He was 79 years of age and a graduate of the University of Toronto (1890).

Dr. George Chauncey Leach, of Toronto, died on April 19, 1937. He was 68 years of age. Born at Roseneath, near Cobourg, he attended Cobourg High School and later the University of Toronto, where he graduated in Arts. Afterwards he attended Queen's University, graduating in medicine in 1904. Dr. Leach then went to Peterborough, where he practised for four years. Following his stay in Peterborough, he practised in Hamilton, working in the latter city for 22 years. A little more than 2 years ago he opened a practice in Toronto.

Dr. J. H. MacDonald, M.O.H. for Ingersoll, Ont., met death on April 26, 1937, by a most regrettable accident. He was speeding along eastbound on No. 2 highway in order to give help to those suffering from flood conditions in the district about London, and when crossing a bridge near Beachville, was swept off into the water. His loss will be much felt.

Dr. James Blainie Hall McClinton, of Midland, Ont., died on April 12, 1937, in his 79th year. He was born in Mulmur Township and graduated from Trinity University, Toronto, with his medical degree in 1888. He practised at Elmvale until 1916, when he suffered a breakdown in health, and later moved to Midland, where he was in practice until recent years.

Dr. Samuel Harvey McCoy, of Ottawa, died on April 9, 1937, aged 72.

Born at Hamilton, Ont., on May 13, 1864, Dr. McCoy was a son of the late John McCoy and his wife, Elizabeth Jane Scott. He received his early education at Hamilton, later attending the University of Toronto where he received his B.A. degree in 1889 and M.D. in 1892.

Upon graduation he practised at St. Catharines, where he was in charge of Welland House Spa until 1903, when he went to England, doing post-graduate work for four years. He was at University College Hospital, London, and at various hospitals on the Continent during that time. He also received his F.R.C.S. at Edinburgh University.

Returning to St. Catharines, he resumed practice there until 1910, when he went to Toronto, where he followed his profession until 1915. In that year he was appointed to the medical staff of the Canadian Expeditionary Forces, having already to his credit 19 years of service as surgeon with the 19th Lincoln Regiment, non-permanent militia. Going overseas, he assumed his duties as a member of the permanent medical board in London, and while there was also attached to the Petrograd Hospital and to the Perkins Bull Hospital.

Returning to Canada in 1919 with the rank of Lieut.-Colonel, he came to Ottawa where he became especially employed on the compilation of medical data in connection with the hospitalization of personnel who had served overseas in the Great War. These data were embodied in the Canadian Government's official history. Dr. McCoy left the militia in 1921, and since then had practised surgery, specializing in women's diseases.

Dr. A. Lapthorn Smith, formerly well known in Montreal, died in England on April 6, 1937, and was buried in Mount Royal Cemetery, Montreal.

A prominent member of the medical fraternity for nearly half a century, Dr. Smith was born in Saint John, N.B., on June 6, 1865, a son of William Smith and Jane Cater Busby. He received his early education in Ottawa City, graduating at the age of 21 years. Going abroad, he continued his studies at Guy's Hospital, London, where he obtained his M.R.C.S., and at the clinics of Vienna and Berlin. He then returned to Montreal to establish practice. During his years in Montreal Dr. Smith became widely known. One of the founders of the Samaritan Hospital, he was its chief surgeon. For many years he was gynæcologist to the Western Hospital, professor of gynæcology and abdominal surgery of Bishop's College University Medical School, later absorbed by McGill University, and visiting lecturer in gynæcology to the University of Vermont.

During the Great War, Dr. Smith served with the R.A.M.C. as chief-surgeon of the Countess of Weymss' Hospital at Chateau Fayel in Compiegne, and later was in charge of the Officers' Hospital, Aldershot. After the war he settled in London and took up practice, first in the west end, but later established himself in south-east London where he worked among the poor until 1923. At this time he was forced to retire on account of ill-health, and until the time of his death lived in retirement at Virginia Water, Surrey.

He is survived by one daughter, Mrs. A. V. Bunting; four sons, Cecil G. Smith, of Montreal; Arthur B. Smith, of Toronto; Alex. B. Smith, of Fort Saskatchewan, Alta., and Ernest L. Smith, at present residing in the United States. His wife, the former Jessie Victoria Buntin, predeceased him some years ago.

Dr. Frank Ware Smith, of St. Thomas, Ont., an eye, ear and nose specialist, died on April 24, 1937.

Dr. Smith was born near St. George, about 70 years ago. Following his graduation from the University of Toronto (1894), he practised in Aylmer for a time and later in Calgary. He returned east from Calgary in 1914 to establish his practice in St. Thomas.

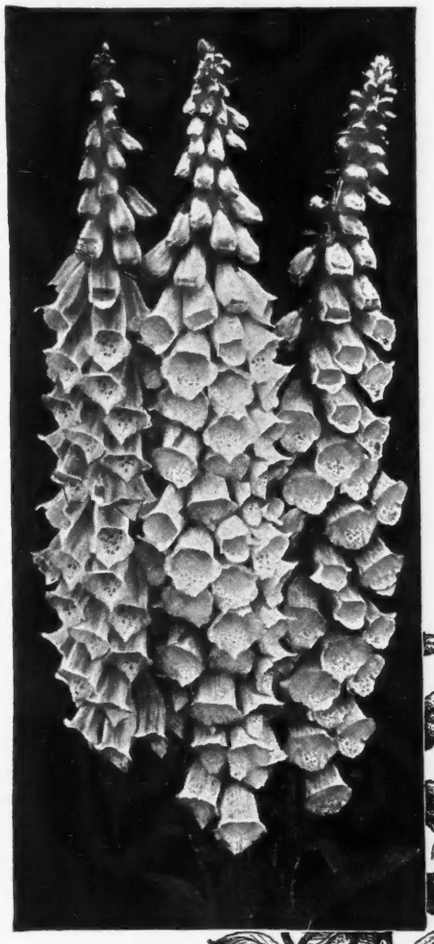
Dr. John Francis Ten Eyck, a practising physician in Toronto for nearly 50 years, died recently in his 74th year. He was the son of the late Mr. and Mrs. John Glover Ten Eyck, United Empire Loyalists. He was born in Binbrook, Ont., and received his education at Grimsby Public School and Hamilton Collegiate, graduating in medicine from Trinity University, Toronto (1899).

Dr. J. M. Trudeau, of Montreal, died on May 1, 1937, in his 69th year. He was born in St. Louis de Gonzague, completed his classical course at the Montreal College and his medical studies at Laval University in Montreal (1894). He was later attached to Notre Dame Hospital, and was a member of the Cercle Universitaire.

Dr. James Benson Whitely, of Goderich, Ont., died on April 12, 1937, aged 81 years. He attended the Goderich Collegiate Institute and graduated in medicine from Victoria University (1883). He had been in practice nearly 54 years. He had been M.O.H. for Goderich for more than 50 years.

Dr. Cleveland Roy Wilson, of Hamilton, an eye, ear and nose specialist, died on April 19, 1937.

Dr. Wilson was the only son of the late William Wilson, first principal of Annette Street School. He graduated with honours from the University of Toronto in 1911, and practised in Toronto until he went overseas as a captain in the Canadian Medical Corps. After the armistice he continued his medical studies in England, Wales and Vienna, and on his return to Canada opened a practice in Hamilton, where he remained until he was taken ill.



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Literature on request

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News Items

Alberta

At the request of the Alberta Federation of Women, a conference has been called by the Minister of Health, Hon. W. W. Cross, to meet in his office, Edmonton, on May 21, 1937, to discuss the various aspects of the subject of birth control. Dr. W. A. Wilson, of Edmonton, President of the College of Physicians and Surgeons, will represent the medical profession.

The Provincial Travelling Health Clinic will commence its work this year on May 16th, and it is calculated that it will be on the road for two months. As in former years, it is planned to cover districts where physicians are not available. Dr. Edward Hitchin, a graduate of the class of 1934, University of Alberta, will join the staff this year. He has been senior intern in the University Hospital for the past year and has been on the staff since graduation.

The Hospital Board of Lacombe is contemplating the erection of a new and larger hospital in that town. Architects are already working on the plans. When completed the hospital will have 30 beds.

Recently the Hospital Board at Carmangay has acquired the former premises of the Bank of Commerce, and the building is being fitted up as a hospital. It will be larger by far than the present one and has excellent lighting and heating arrangements.

The District Associations in Alberta are having their meetings in consecutive order this year, which makes it possible for the President-elect, the Honorary Secretary and clinicians to make the tour without being too much away from their practices. It is expected that the annual elections will take place at these meetings. The Board of Directors was called to meet in Edmonton on June 2nd, when a closer relation between the College and the Provincial Medical Association was up for discussion.

A special meeting of the Council of the College of Physicians and Surgeons will be held in Edmonton, June 3rd next, when routine business will be taken up.

Owing to the unsettled condition of the political situation in Alberta, no legislation seriously affecting the medical profession, has been so far undertaken by that body, but what they will do at the adjourned session in June is still unknown. G. E. LEARMONTH

British Columbia

Premier T. D. Pattullo has announced that a general election will be held on June 1st, and at this time the voters will be asked to express an opinion on Health Insurance. A referendum will accompany the election ballots with the wording "Are you in favour of a comprehensive Health Insurance plan, progressively applied?" The premier has signified that if the referendum receives an affirmative answer health insurance will have to start in a "limited way", which is understood to mean that the previous Health Insurance Act will be scrapped completely. In an advertisement in the daily press throughout the province the government's endorsement of a health insurance bill has been reiterated. At the same time it is stated that the care of indigents will be considered as a separate question and not included in any new bill. It is thus apparent that the government is resolved to ignore what is considered to be one of the chief objections brought against health insurance legislation by medical and many other opponents as proposed or enacted in the past. Nevertheless the government candidly admits that it was unable to cope with the overwhelming weight of protest which the recently passed Health Insurance Act produced.

An editorial in the *Victoria Daily Colonist* of April 16th strongly criticizes the proposed plebiscite. It states the "somewhat bald question still leaves the matter up in the air. Is the insurance spoken of to be compulsory or optional? Are the plans for it to be made by the same authorities as conceived the last abortive scheme, or . . . ? The words 'comprehensive' and 'progressive' mean little or nothing. . . . The last plan, which is to be wiped off the statute books, claimed also to be 'comprehensive' and 'progressive'." The editorial goes on to say that the people "are asked to commit themselves one way or another on the subject of health insurance without the slightest inkling of what is projected, and having the experience that after a long period of intensive research work and more intensive propaganda the Government failed to devise a workable scheme".

On Coronation Day the Lady Elizabeth Bruce Memorial Hospital at Invermere will be formally opened. The building is the former residence of Hon. R. Randolph Bruce, Canadian Minister to Japan, and Lieutenant-governor of British Columbia from 1926 to 1931. Hon. Mr. Bruce has met all expenses in connection with converting the building, beautifully situated on Lake Windermere and surrounded by handsome gardens into a thoroughly modern and fully-equipped hospital, as a permanent tribute to the memory of his first wife, Lady Bruce, the former Lady Elizabeth Northcote, who died in 1915. Dr. F. E. Coy will be medical director and Miss M. Thatcher, R.N., will be matron-superintendent, with Miss M. Peterson, R.N., as her assistant.

Dr. A. D. Lapp, superintendent of Tranquille Sanitarium, Kamloops, since 1921 has resigned, and has left to reside in eastern Canada. He will be succeeded by Dr. H. S. Stalker, assistant superintendent of the Vancouver General Hospital.

Dr. R. A. Seymour, at present on the staff of the Mental Hospital at New Toronto, is returning to resume the position which he formerly held as assistant superintendent of the Vancouver General Hospital, which he left in 1930 in order to take up special training in eastern Canada. D. E. H. CLEVELAND

Manitoba

The following additions have been made to the Honorary Attending Staff of Grace Hospital, Winnipeg: Drs. C. B. Stewart and D. Schwartz, Urologists; G. H. Ryan and A. E. Deacon, Orthopædists; Anne E. Wilson, Pædatrician; A. T. Gowron, Anæsthetist.

The Winnipeg General Hospital has recently installed a vocal call system throughout the hospital.

The first of a series of monthly clinical meetings of the Honorary Attending Staff of the Winnipeg General Hospital was held in the Board Room of the hospital on April 27th when the following program was presented: Dr. N. W. Warner, "Adult scurvy": Dr. C. R. Gilmour and Dr. W. Boyd, "Carcinoma of the liver"; Dr. M. R. MacCharles and Dr. Wm. Boyd, "Brain tumour". Dr. D. C. Aikenhead presented a report from the Department of Anæsthetics.

A meeting of the panel practitioners of Winnipeg was held in the Medical College on April 29th. The use of the new card system for sick unemployed was demonstrated with lantern slides. There was a free discussion of the panel system as it is met with in Winnipeg.

CHOLELITHIASIS, cholecystitis, any recurrent gastric irritation may produce direct or reflex localizing symptoms. Or again, symptoms may be of an obscure and indefinite nature, presenting a complicated diagnostic problem. And since early treatment is important—oftentimes imperative—the information disclosed by radiography of the gall-bladder is invaluable.

Of the available diagnostic measures, none has so conclusively proved its accuracy and importance as cholecystography. This radiographic method alone definitely demonstrates functional characteristics of the gall-bladder . . . reveals practically all types of calculi . . . discloses anatomical relationships to contiguous structures . . . differentiates between papilloma and diverticulum.

Especially where symptoms are vague, where the patient has experienced no characteristic biliary colic, is thorough x-ray examination by a radiological specialist advisable. Canadian Kodak Co., Limited, Toronto, Ontario.

RADIOGRAPHS PROVIDE DIAGNOSTIC FACTS

On May 15th, Dr. A. B. Alexander, having reached the age of 65, retired as Superintendent of Winnipeg's municipal hospitals, a position which he has held for over twenty-five years. He is succeeded by Dr. Dugald McIntyre, who has been Assistant Superintendent.

The sixtieth anniversary of the founding of the University of Manitoba was observed at the Convocation in May.

Dr. A. J. Douglas, the veteran Health Officer of the City of Winnipeg, received the honorary LL.D. degree.

The address at this session of convocation was given by Vilhjalmar Stefansson, the noted Arctic explorer, (born in Manitoba), who also was granted the honorary LL.D. degree.

ROSS MITCHELL

New Brunswick

Dr. W. W. White, of Saint John, was recently in Ottawa. He was invested on April 20th by Lord Tweedsmuir, representing His Majesty, King George VI, with the emblem of his rank as an officer of the Order of St. John of Jerusalem.

Dr. R. J. Collins, Superintendent of the Saint John Tuberculosis Hospital, it at present in England, doing post-graduate study.

Under the direction of Dr. Charles MacMillan, local health officer, the school children in Saint John, who have not previously been protected against diphtheria are being inoculated with toxoid. An opportunity for all pre-school aged children to attend these clinics at the same time, is being provided.

Drs. A. B. Walter and H. A. Farris of Saint John, recently attended the meeting of the American College of Physicians, at St. Louis. Dr. Farris was elected Governor of the College at this meeting.

Hon. Dr. W. F. Roberts, Minister of Health for New Brunswick, has just returned from a visit to Montreal, Toronto, Boston and New York, at which points he made departmental inquiries as to the value of physical medicine in the handling of Workmen's Compensation cases. Other matters of public health nature were also investigated by the Minister.

Dr. W. E. Rowley is again confined to the hospital, suffering from a cardiac lesion.

The many friends in the Canadian Medical Association throughout Canada, of Dr. G. Clowes VanWart, of Fredericton, will be glad to hear that he is making satisfactory recovery from the serious accident which he suffered last winter.

A. S. KIRKLAND

Nova Scotia

Drs. J. G. MacDougall and H. K. MacDonald, of Halifax, appeared before the Law Amendments Committee of the Legislature on behalf of the Nova Scotia Medical Society, to urge removal of clauses in the Workmen's Compensation Act which limits medical aid to an injured workman to thirty days. They also opposed the suggested reduction from 75 cents to 50 cents for travelling rates per mile. Dr. MacDonald had previously appeared before the Royal Commission investigating operation of the Workmen's Compensation Act, and had proposed to that body too that the limit of thirty days for medical aid be abolished.

The Halifax Curling Club devoted the proceeds of its fourth charity bonspiel to the Halifax branch of the Victorian Order of Nurses. A sum of approximately $550 was raised to help this organization in its work.

The board of directors of the General Hospital at New Waterford, has approved an eight-hour day for nurses as from May 1st. The General Hospital has also installed a complete new x-ray equipment. The advisability of securing the services of a radiologist exclusively for Cape Breton will also be discussed with other institutions.

Dr. P. S. Campbell of the Public Health Department, has visited North Sydney to examine the Federal Air Station Buildings which have been suggested as an extra asylum for Cape Breton. The report suggests that the Joint Expenditure Committee reconsider the whole matter before embarking on the scheme, as several difficulties arise in converting these buildings to hospital use.

Opposition to the erection of a new hospital at Kentville has been voiced by representatives of the West King's Hospital at Berwick and of the hospital at Wolfville. The nearest hospital to Kentville is seven miles distant.

The 12-bed hospital put up five years ago at Cheticamp is to be replaced by a new building, capable of accommodating 40 patients. Work has already begun, and it is expected that the hospital will be ready for occupation in December.

Lieut.-Col. O. G. Donovan, medical officer for Military District No. 6, and recently appointed on Nova Scotia's section of the Canadian Coronation Contingent, has been advised by Ottawa that he has been appointed a staff officer on the Contingent in charge of all medical arrangements.

N. B. DREYER

Ontario

Dr. Samuel Hamilton, New York psychiatrist, has presented to the Provincial Government his report on the survey of mental institutions in this province. Doctor Hamilton was loaned for this purpose by the Carnegie Institute. The report has not yet been printed for distribution, so far as we can ascertain, but from newspaper reports we learn that among the recommendations are the following: Better pay and shorter hours for nurses; twenty-four more doctors; a school for male nurses; the removal of the tuberculous insane from the special unit of the New Toronto Hospital to a new and specially designed plant; that the government should consider the question of mechanical additions, that is window guards, closed wards and so forth; that they temporarily agree to immediate admissions, provisional quarters to be arranged for 1,000 patients, perhaps in some fireproof factory or similar structure; that there be closer co-operation between the public schools and mental institutions, so that feeble-minded and insane children might be examined and given a place.

He suggested a complete program to be carried out within the next seven years which would include a hospital near Toronto for 500 tuberculous insane, and 1,000 infirm patients; a 1,200-bed hospital at Fort William, and at Woodstock, 400 new beds. There were many recommendations as to the enlarging of the present accommodation at Woodstock, Kingston, Brockville, London, Toronto Pyschiatric Hospital, Penetanguishene, New Toronto, Orillia, and Cobourg. Among other suggestions, he states that the libraries provided

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for patients are often very poor and that more books should be available and the library should be in charge of someone with library training. The report considers the treatment of alcoholism and drug addiction. Throughout the report, Doctor Hamilton makes many references to the over-crowded conditions existing in the present hospitals.

The report, when published, should offer material for thought for those who are interested in the problem of mental health. It is apparent that the government is wide awake to the necessity for the improvement of the hospitals. This report should strengthen them in their proposals for many desirable changes.

Before the tabling of Doctor Hamilton's report, an announcement was made by Dr. B. T. McGhie, Deputy Minister of Health, that 70 patients would soon be moved from the New Toronto hospital to the new tuberculosis ward at Concord which is being fitted for their reception. The Minister announced that all nurses caring for tuberculous patients will henceforth receive $1,125.00 per year.

The Premier announced in his budget speech that the government plans, within the next fiscal year, to spend a considerable sum of money for additions to government-owned hospitals and on new construction.

On the occasion of his retirement from the St. John Ambulance Corps, Dr. Charles Copp, of Toronto, was presented with a "Book of Memories" containing numerous letters of appreciation of the outstanding service he has rendered to the Corps. Doctor Copp was Assistant Commissioner for Canada from 1911 until 1934, when the headquarters of the Canadian Commandery was established at Ottawa.

Dr. Norman M. Keith, head of the section in the division of medicine at Mayo Clinic at Rochester, Minn., and associate professor of medicine at the University of Minnesota's graduate school was the chief guest speaker at the third annual services of post-graduate lectures arranged under the combined auspices of McMaster University and the Hamilton Academy of Medicine on March 30th and 31st.

A hospital is being built at Hawk Junction toward which grants have been made by the Dominion Government, the Ontario Government and the Red Cross Society. At its inception the hospital will have eight beds and will be under the direction of the Red Cross Society. It will serve the Wawa district and the Goudreau mining area as well as the section immediately around Hawk Junction.

Dr. A. E. Young has been appointed to the Public Health staff of the City of Toronto, to fill the vacancy caused by the death of Dr. R. Challener.

The Second Annual Hospital Day of the Hamilton General Hospital was held on April 28, 1937. There was an attendance of 350 medical men representative of some 80 points outside of the city. The day was entirely devoted to clinical demonstrations of various procedures useful to the general practitioner and no didatic lectures were presented.

Scientific exhibits were presented by various departments, including pathology, metabolism, diabetes and hæmatology.

Interesting displays of some 35 commercial exhibitors also added to the success of the meeting.

The visiting doctors were guests of the Hamilton General Hospital at a buffet luncheon during the course of which they were welcomed to the institution by Mr. W. H. Cooper, President of the Board of Governors and by Dr. H. W. Whytock, chairman of the Medical Staff.

The Trustees of the Listowel Memorial Hospital have prepared plans for a new wing at a cost of $40,000. Perth County Council has made a grant towards the erection of the new wing which will be paid over a period of four or five years. The increasing demands upon the hospital are indicated by the fact that in 1929 the number of patients admitted was 350; in 1933 there were 303; and in 1936, the total was 475.

On April 13th, Dr. J. E. Mabee, of Odessa, who celebrated his fiftieth anniversary in the medical profession, was the guest of honour at a dinner held by the Kingston and Frontenac Medical Society at the Kingston General Hospital. A large number of the doctors of the district were in attendance to pay tribute to their colleague who still continues his practice. A large birthday cake with fifty candles stood in the centre of the table. A number of brief addresses were given, paying tribute to Dr. Mabee, after which he was presented with a gold-headed cane, the presentation being made by Dr. L. J. Austin. Dr. J. S. Delahay presided at the banquet. In his reply, Dr. Mabee expressed his deep gratitude to his colleagues for their kindness. He then entertained the audience with some humorous stories of happenings in the early years of his practice. J. H. ELLIOTT

Quebec

Dr. Guy H. Fisk, of Montreal, a graduate of McGill University (1933), has been awarded the diploma in tropical medicine and hygiene of the Royal College of Physicians and Surgeons of England. He sailed to take up a post as medical officer in the Colonial Medical Service in Nigeria.

Saskatchewan

Dr. James C. Paterson has been appointed pathologist to the Regina General Hospital. He graduated from the University of Toronto in 1925. He was with the International Petroleum Company in Peru and Colombia for nine years. He held a Fellowship in Pathology and Bacteriology for two years at the University of Toronto, working on the etiology of coronary thrombosis. His preliminary report was published in the *Archives of Pathology* in September, 1936. LILLIAN CHASE

United States

The American Board of Ophthalmology. — The American Board of Ophthalmology has decided to establish a preparatory group of prospective candidates for its certificate, the plan being to assist physicians who wish to study ophthalmology so that they may become acceptable as candidates for examination and certification when they have completed the requirements.

Any graduate or undergraduate of an approved medical school is eligible to make application for membership in this group. Candidates so applying will be notified officially by the Secretary when the Board has accepted their applications. If accepted, data will be sent concerning ethical and educational requirements. Syllabuses and other information will be made available to them.

Information and advice will be available to members in this group through preceptors who are members or associates of the Board. Members of the preparatory group must keep a summarized record of their activities, two copies of which will be sent to the

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Although not epidemic in Canada, typhoid and paratyphoid infections remain a serious menace—particularly in rural and unorganized areas. This is borne out by the fact that during the years 1931-1935 there were reported, in the Dominion, 12,073 cases and 1,616 deaths due to these infections.

The preventive values of typhoid vaccine and typhoid-paratyphoid vaccine have been well established by military and civil experience. In order to ensure that these values be maximum, it is essential that the vaccines be prepared in accordance with the findings of recent laboratory studies concerning strains, cultural conditions and dosage. This essential is observed in production of the vaccines which are available from the Connaught Laboratories.

Residents of areas where danger of typhoid exists and any one planning vacations or travel should have their attention directed to the protection afforded by vaccination.

Information and prices relating to Typhoid Vaccine and to Typhoid-Paratyphoid Vaccine will be supplied gladly upon request.

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secretary in January of each year and will be incorporated in the final application for examination and certification.

The fee for application for membership in the preparatory group will be $10.00. This will be deducted from the $50.00 which is required of every candidate for examination.

Important notice.—The American Board of Ophthalmology will conduct an examination in Philadelphia, June 7, 1937, and in Chicago, October 9, 1937. All applications and case reports, in duplicate, must be filed at least 60 days before the date of examination.

John Green, M.D., Secretary,
3720 Washington Blvd.,
St. Louis, Mo.

General

The Fourth International Pædiatric Congress is to be held in Rome from September 27 to 30, 1937. The following subjects will be discussed:—Mineral and Water Metabolism in Infancy, Tuberculosis in Childhood, Neuropsychiatric Disorders in Pædiatrics from the Clinical and Social point of view.

Those interested and requiring further information are invited to communicate with Dr. H. P. Wright, 1509 Sherbrooke Street, West, Montreal, secretary of the Canadian Committee for the Fourth International Pædiatric Congress.

Book Reviews

Eugenical Sterilization: A Reorientation of the Problem. By the Committee of the American Neurological Association for the Investigation of Eugenical Sterilization. 211 pages. Price $3.00. Macmillan's, Toronto.

This book is a fairer statement of the problem than would be expected when the Committee was headed by a chairman who has said that the idea that mental conditions are inherited is now going the way of all other long discarded superstitions, and that he has to draw upon his hopes as well as facts in order to produce evidence that adverse environmental conditions are responsible for transmissible mental diseases.

The laws which have been passed for sterilization; the main arguments and viewpoints concerned with this question; the relation of genetics, which is the straight science of inheritance, to eugenics, the social science; of sterilization to crime and to genius; the inheritance of schizophrenia, manic-depressive psychosis, feeblemindedness and epilepsy, and the importance of twins as material for genetic study are discussed, followed by the recommendations for sterilization advocated by the committee. With these, the great majority of those in favour of eugenics will agree; namely; (1) that the consent of the patient or his guardians be obtained for the operation; (2) that the law for sterilization apply not only to those in State Institutions but to those in private institutions and to those at large in the community. This last provision is of course what eugenicists are striving for. So long as the patient is an inmate of any institution he or she is effectively sterilized by mere isolation. It is for the applicability of the procedure especially to those who are in a position to procreate that the eugenicist strives. The committee advocates the establishment of a Board with training to review the cases for whom sterilization is proposed, and the protection of such a Board and the surgeon by legal enactment. The diseases which they feel call for sterilization are listed in the order in which they feel there is evidence for heredity as the cause. They are Huntington's chorea, hereditary optic atrophy, familial cases of Friedreich's ataxia, and certain other disabling degenerative diseases recognized to be hereditary, feeblemindedness of familial type, dementia præcox, manic-depressive psychosis and epilepsy.

They recognize that there may be a social as well as a biological foundation for sterilization, and that a patient even with a non-hereditary type of feeblemindedness is scarcely the person to be the parent of a normal child who must depend upon such inadequate parentage for care, food and shelter. Finally, they urge with respect to the manic-depressive psychoses a valuation of the entire assets of the individual before advocating sterilization. Finally, the Committee calls for a program of research, which we sorely need not only from the standpoint of finding what part heredity plays in the production of these diseases but equally for the accurately controlled determination of what part environment plays. Just as we do not want to launch upon a program of wholesale sterilization of mental patients without adequate knowledge of the inheritability of mental disease so we should be just as wary of expending millions upon altering the environment in the hope of eliminating mental diseases, until we have proved definitely that such alteration will accomplish the desired result. The committee are unduly optimistic when they feel that a decade of investigation will make an appreciable impression upon the problem.

Historical Notes on Psychiatry. J. R. Whitwell, M.B., Honorary Librarian, Royal Medico-Psychological Association. 252 pages. Price 10s. 6d. Lewis, London, 1936.

This monograph fills a need long felt by those interested in the historical side of medicine, and makes available material which heretofore has been difficult to obtain. Dr. Whitewell writes well on a subject with which he is fully qualified to deal, and what few comments he makes are acceptable to modern psychiatrists. He summarizes briefly, yet thoroughly, the theories and writings of the famous and less famous medical writers from the earliest times to the end of the sixteenth century. Direct translations are given where advisable for the sake of clearness.

The book contains nine chapters, the first of which deals briefly with the writers of the ancient period in China, India, Babylonia and Egypt when, "out of the dim mists of antiquity, against a background of primitive superstition, there appears from time to time, here and there, an outstanding figure, earnest, sincere and clear thinking, striving to fathom the eternal problem of nature, to unravel the mysteries and secrets of science". The Scriptures are considered in the second chapter; the Classics in the third; observations of the Greek philosophers in the fourth, while the classical medical writers are given fuller consideration in the fifth chapter. The sixth chapter reviews the then recognized mental disorders, and for that reason is of particular interest. The seventh chapter deals adequately with the middle-age period. Chapter eight is noteworthy in that here the author gives translations and extracts in chronological order from the manuscripts of 65 medical writers, ranging from Hippocrates to Barrough. The present source of this material is indicated. The final chapter is a chronological table of those contributions which in the opinion of the author are of value in understanding the evolution of modern psychiatry. The author's excellent arrangement of his material has obviated the necessity for an index. This book will prove interesting to all physicians and of definite value to medical historians and psychiatrists.

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Original Papers of Richard Bright on Renal Disease. A. A. Osman, D.Sc., F.R.C.P. 172 pages, illustrated. Price $6.30. McAinsh, Toronto, 1937.

There is an increasing number of physicians keen to read original descriptions of disease. Richard Bright's four publications have long been out of print. Bright, like Laennec, was a master of observation, keen to observe and clear in his descriptions. His "Reports of Medical Cases" must be looked upon as one of the landmarks of medical progress. In this present volume Dr. Osman has reprinted it page for page, with the five magnificent coloured plates. He also reprints three other papers by Bright dealing with renal disease. Bright made many other important contributions to medicine, but this volume deals only with those relating to the kidneys. In an appendix of 6 pages Dr. Osman describes the gross and microscopic appearances of the tissues of three of Bright's specimens preserved in the Museum at Guy's Hospital. A clinical teacher will be very happy to possess this excellent reprint of classical British contributions to medical science.

The Operations of Surgery. R. P. Rowlands, M.S., F.R.C.S., and Philip Turner, B.Sc., M.S., F.R.C.S. Eighth ed., vol. 2, The Abdomen, 228 pages, illustrated. Price $10.50. Macmillan, Toronto, 1937.

The review of the first volume of this well known work appeared in the September, 1936, issue of the *Journal*. The second volume maintains the high standard of excellence which has characterized the former editions.

An extensive re-writing and revision of many of the chapters has been made by Mr. Philip Turmer, assisted by Mr. W. H. Ogilvie, Mr. A. R. Thompson, and Mr. G. F. Libberd.

A new feature which has been included, is a chapter on "Some recent developments", which it is hoped will be "a useful guide to some of the new methods of operative technique and their clinical applications". For a period of 48 years this treatise has been a standard work on operative technique in Great Britain. Down through the years the editors have kept it abreast of the times. Though it is representative of British surgical technique, yet due credit is given to the opinions of both American and Canadian surgeons. This volume is a worthy successor of the former ones. It is noteworthy for the soundness of surgical opinion as well as for clarity of expression. The illustrations are good and quite numerous. This excellent volume requires no further commendation.

An American Doctor's Odyssey. Adventures in 45 countries. Victor Heiser. Price $3.50. McAinsh, Toronto, 1936.

It would be much easier to write a review of this book in a very few lines than in just a moderately few. To begin with, it covers a long period, opening with a personal experience in the Johnstown flood in 1889. Then, it deals with the experiences of a very active medical man throughout an era of preventive medicine dramatic in the highest degree. Finally, it is written with a great deal of detail. Dr. Heiser has a curiously detached manner. Even when he floats into our ken on the bosom of the Johnstown flood he makes it seem interesting rather than tragic. He becomes most nearly violent when he criticizes the political interference which removed, in the person of W. Cameron Forbes, one of the finest governor-generals in the history of the Philippines (and is there any form of human folly more painful than political meddling?). He goes from place to place, and disease after disease is picked up and described, until we have as good a history of the chief tropical diseases as most people need to have, and a great deal more interesting than many other such histories.

The index is poor. It might have been better to make a name index alone. But even a mere list of the names in this book would be impressive. The author chose his title well. It is an art, which he has freely exercised, to be able to convey so lively and attractive a sketch of his very full life.

Plastic Surgery of the Nose. J. E. Sheehan, New York. Second ed., 186 pages illustrated. Price $9.00. Hoeber, New York, 1936.

This edition is entirely rewritten. The table of contents shows, Anatomy and Function, Typical and Distinctive Operations, Classified Corrections at the Three Nasal Levels, Skin Grafts, Precaution, Preparation and Postoperative Care. The book is well illustrated and the author has gone to considerable trouble to outline the anatomy and function of the nose and associated parts. The description of distinctive operations is detailed and clear. He describes a number of operations which appear to give excellent results, but which in the hands of many others have not been as satisfactory as outlined in this monograph. It is interesting to note that for support he has not changed from the old accepted principles, namely, the use of cartilage instead of the use of bone, as advocated by some surgeons. A chapter is given to mechanical restoration which should be helpful, but too much confidence should not be placed in the use of mechanical support for the nose. It is interesting to note the author's meticulous care in describing minor operations for certain corrections.

This is a book which should be read by all men doing reparative surgery, and one which perhaps will be of more benefit to the specialist than to the general practitioner, but it is a worthy contribution to reconstructive surgery.

Urological Roentgenology. M. B. Wesson, M.D. and H. E. Ruggles, M.D. 269 pages, illustrated. Price $5.00. Lea and Febiger, Phila., 1936.

This handbook, while written, as the title states, for the general practitioner, will be a welcome addition to the library of everyone interested in this subject.

The volume covers, in very readable manner, history, technique and diagnosis. The discussion and evaluation of the retrograde and intravenous methods are most timely. Almost every type of pathological changes and also congenital anomalies are discussed in a sane and candid manner. The illustrations are exceptionally clear, and it is a pleasure to have these done in the negative phase, which makes it particularly useful to the student. There is also an excellent bibliography, well selected, and up to the minute.

Blood Cultures and their Significance. Hildred M. Butler, B.Sc., Bacteriologist, Baker Institute of Medical Research, Alfred Hospital, Melbourne. 327 pages. Price 15s. Churchill, London, 1937.

This monograph from the Baker Institute will be a most welcome addition to the library of every bacteriologist and clinician. In this concise, but nevertheless reasonably complete, account of the subject the author has endeavoured, very successfully, to show the importance of bacterial cultivation of the blood in human disease and to indicate the best ways to eliminate false negative results.

The book is divided into 20 chapters, the first three dealing with the general aspect of the subject, the remainder devoted to consideration of the nature, duration and significance of bacteriæmia in various diseases and to special methods of cultivation. Practically in every chapter reviews of the work of other workers are briefly given. By the methods described the author has increased the percentage of positive results about threefold. It is hoped that this volume will be the means of greatly expanding the use of blood cultures in the investigation of disease.